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A

Manual of Medicine

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VOL. III.

DISEASES OF THE NERVOUS SYSTEM

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THE GENERAL ANATOMY AND PHYSIOLOGY OF THE NERVOUS SYSTEM

Of the collection of cells in the embryo that ultimately form the nervous system two kinds may be distinguished. One kind becomes the supporting elements, and in their adult form are known as neuroglia cells. The others become those known as nerve cells proper or neurones. All the discharging and conducting elements of the nervous system are its neurones; and of all its elements only its neurones do discharge or conduct.

The neurone as a transmitter.—Each neurone is an eminently excitable cell. The functional waves of change which it is the office of the nervous system to elaborate and distribute move along nothing else than a concatenation of neurones. As would be expected in links of a chain, the length of each neurone lies parallel with the direction of transmission of impulse. At its one end each neurone is an eminently receptive cell, and easy of access by vibrations impinging on its environment; each neurone is throughout from end to end an eminently conductive cell: that is, it readily propagates a change once started in it. Since one end is receptive and the rest of the cell conductive, the waves of change always travel through the neurone from the receptive end. By powerful artificial means a "change" can be initiated in parts of the neurone remote from the specially receptive end; the "change" is then found to be propagated in all directions along the neurone; but under natural circumstances the "change" is always excited at the receptive end only; hence the direction of propagation is never reversed. The "changes" or "impulses," therefore, circulate in one direction only along the nervous system. This is what has been called by James the "law of forward direction." If the outgoing end of the neurone is turned toward extrinsic—that is, not nervous—tissues, the neurone is *efferent;* if its outgoing end is turned away from extrinsic tissues, the neurone is *afferent.* In being propagated along a chain of neurones, the "impulse" is profoundly and variously modified. This modification occurs perhaps in that

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part of the neurone which contains the nucleus, *i.e.* in the "cell body." It is more probable, however, the modification of the nerve impulse in transmission occurs at the places of linkage of neurones—at the so-called *synapses.* There is, in many cases at least, an intercellular gap between neurone and neurone. Slight contractile retraction of the cell processes at the synapse may afford a means of isolation of one conductor from the next. The inhibitions of hypnosis and the narcosis of opiates have been referred to the opening apart of the nerve-cell *synapses.* It is an established fact that the longer the chain of neurones traversed by an impulse, the more variable and the less predictable the ultimate reaction, and the longer the time occupied by the reaction. The lengthening of time is much greater than the mere lengthening of the path will explain. Each additional synapse introduces therefore a considerable delay in reaction time, and an increase of possible variation in reply.

Minute structure of the neurone.—The *neurone* or *nerve cell* consists of a cell body or *perikaryon*, and of cell branches. The former contains usually one nucleus and nucleolus; but the "nerve" cells of the ganglia of the sympathetic system contain often two, three and even four nuclei. The cell processes are of two kinds, *axons* and *dendrons.* The latter are called *cellulipetal*, because there is evidence that they conduct impulses toward the cell body. The dendrons, as their name implies, are much branched. Many nerve cells possess numerous dendrons; the spinal ganglion cell has, however, only one, and all bipolar cells have only one. The surface of the dendrons of certain cells, *e.g.* the pyramidal cells of the cerebral cortex, is beset with "thorns." The dendrons appear to be fibrillar, and between the fibrils in the stem processes there is a granular material. In the cell body the existence of fibrils separated by granular material is also established (Nissl). The granular material is tingible with methylene blue, and its amount and distribution varies with the condition of the nerve cell; it is used as an index by which to examine the effects of poisons, malnutrition, and retrogressive changes due to disease, etc. Compression of the aorta for a few minutes in the rabbit alters Nissl's granules in the spinal cells. Severance of the dendron not only leads to the degeneration of that process itself but to visible atrophy—probably in most cases only temporary—in the perikaryon itself. Pigment is especially in man a normal constituent of the nerve-cell body. It exists in larger quantity in adult than in young life. It does not contain iron; it is perhaps fatty. It tends to occur in abnormal quantity in diseases associated with excessive nervous activity, for instance in epilepsy and paralysis agitans.

The fibrils demonstrable in the dendron processes are continuous with those in the cell body, but the greater part of these fibrils do not anastomose. They merely sweep through the cell body from one

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dendron to another, or from the dendrons into the axon. In some invertebrata the fibrillæ are continuous not only throughout the whole of one cell and its processes, but exhibit continuity from cell to cell. It is in the axon that the fibrils reach their clearest expression. The axon consists of nothing but a bundle of fibrillæ packed with hardly any intervening substance. The inference is that the fibrillæ indicate lines of conduction in the neurone. The lines of conduction do not necessarily pass through the cell body. The dendron stem may join the axon stalk at a considerable distance from the cell body.

In cells with T-shaped processes the conduction still occurs as usual after the cell bodies have been cut off. The path of conduction, *viâ* the spinal ganglion cell, does not employ the "body" of the cell. Nerve cells, according to the character of the axon, are of two classes: (1) with long axon which is unbranched for a considerable distance; (2) with short axon which breaks up quite close to the cell and soon loses itself in branches. The latter cells are called "link cells" and lie wholly imbedded in gray matter; the former, on the contrary, nearly always thrust the axon as a myelinate nerve fibre into white matter. The myelin sheath must be regarded as a nutrient membrane developed by the nerve cell around the axon, especially where that is large and long. The degeneration of the myelin sheath on severance of the axon from the cell body proves that the myelin sheath is part of the nerve cell. The primitive sheath outside the myelin sheath does not degenerate, and it must be looked upon as a connective-tissue sleeve wrapped round the nerve-cell process where that lies in the non-nervous tissues.

It is of considerable importance that the nerve cells of the body early, very soon after birth, cease to multiply. But they retain remarkable powers of growth and repair. The axon processes if severed degenerate, but are in four or five weeks' time replaced by sprouts from the stumps of the old mutilated processes, and these reproduce in time the myelin sheaths, and attain the size of their predecessors. Some hold that regenerate nerve fibres are formed from the peripheral piece independently of the stump.

In many nerve cells, amputation of the axon not only causes Wallerian degeneration of the severed nerve fibre, but changes in the perikaryon itself. The granules of Nissl become less defined, and the neighbouring portions of the protoplasm diffusely coloured. The cell body then becomes swollen, and the nucleus pushed to one side. In fifteen days' time the cell, instead of being "tigroid," is nearly uniformly stained with the methylene blue. The change invades the cell from the point of exit of the axon from the cell body. It extends gradually into the dendrons. At the end of fifteen to twenty-four days a reparative process sets in, and the cell regains its normal tigroid appearance in about three months. On cutting the vagus trunk chromatolysis and ultimate degeneration and disappearance of the cells of its ganglion

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ensues. Some motor-horn cells disappear altogether in the lumbar region after section of the afferent roots of that region (Warrington). Visible changes ensue on prolonged activity. The perikaryon becomes reduced in size and stains less deeply. The nucleus shrinks.

The root cells and reflex action.—The *nerve impulse* is the unit of function arrived at by physiological analysis of the neurone. Almost all nervous reactions are ultimately reactions to the environment. They are reflex in the sense that the environment is causally related to them, and that to the environment they return again. The nervous system rests upon the two *root cells* as upon an arch. On this arch are superposed many others of greater and less complexity, but the prime pillars are those of the basal arch. These pillars are the *afferent root cell* and the *efferent root cell.*

The *afferent root cell* conveys impulses, generated by the environment, into the central nervous system. The central nervous system derives its right to be sundered from the rest or peripheral nervous system in virtue of its containing all the synapses (places of linkage) of the entire nervous system. The environment usually excites the afferent root cell only mediately. That is, its direct action is upon non-nervous cells, which in their turn act upon the peripheral end of the afferent root cell. The cells thus surrounding and acting on the root cell form, together with the ends of the root cell they contain, a sense organ, *e.g.* tactual corpuscle, cochlea, outermost layers of retina, etc. In a few cases, the end of the root cell lies directly accessible to the environment, *e.g.* the "pain" nerve endings in the cornea, the olfactory cells in the Schneiderian membrane. In most the mediate cells which transfer the environmental change to the nerve cell, are epithelial; but in some, *e.g.* muscle spindles, Pacini corpuscles, Golgi's tendon organs, etc., they are mesoblastic, such as muscle fibres, connective tissue, etc.

As a rule the afferent root cell has its cell body outside the central nervous system in the ganglionic masses on the afferent roots of the spinal and cranial nerves. An exception occurs in the olfactory path. There the bodies of the afferent nerve cells lie in the same primitive condition as in the skin of the earthworm, scattered up and down the Schneiderian epithelium, the long, deeper processes extending as true nerve fibres into the olfactory lobe. Again, in the eye the afferent root cells lie spread in a single layer, the inner nuclear layer of the retina.

The afferent root cells may be usefully considered in brief as follows:—

(1) *Olfactory*, probably acted upon directly by chemical agents dissolved in the film of fluid moistening the olfactory mucous membrane.

(2) *Visual*, acted upon by radiant energy indirectly, probably through chemical changes in the rod and cone epithelia.

(3) *Auditory*, a cochlear set and a labyrinthine set. The latter are

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the more primitive, the former being possessed by the higher vertebrata only. The labyrinthine furnish perceptions of the position of the head in space, and so of the whole body, since the spatial relations between the head and body are relatively constant. The cochlear set subserves the sense of hearing; the external stimulus for both is of mechanical quality.

(4) *Gustatory*, the stimuli are chemical. The nerves are the third division of the fifth cranial nerve, the chorda tympani branch of the facial, and the glossopharyngeal.

(5) *Tactual*, excited by any agent which deforms the normal contour of the skin surface. The tactual afferent root cells are distributed in a patchy manner, so that analysis of the skin by minute stimuli reveals areas whence tactual sense is absent. Tactual nerve fibres lie in the afferent roots of the fifth and ninth cranial, and of all the spinal nerves except the first cervical.

(6) *Dolorous*, of very wide distribution, in the skin, viscera, muscles, joints, and certain skin appendages, *e.g.* teeth, cornea. In the skin "pain-spots" are more densely set than are the tactual. The mucosa of the inside of the cheek is in places completely devoid of them. The afferent root cells which are concerned with sensations that are referred to the body itself, *e.g.* its muscles, viscera, skin, etc., all seem under "excessive" stimulation to provoke "pain." Such nerves are known as nerves of "common sensation."

(7) *Thermal*, divisible into those conducting from *cold spots* and those conducting from *warm spots*. The former are the more numerous, but not so numerous as the tactual. They are dotted over the skin surface generally, and over the mucosa of the mouth. The adequate stimulus seems to be rapid alteration of skin temperature.

(8) *Muscular*, already referred to under dolorous, because they can excite pain; they are root cells that end peripherally in muscle spindles, tendon organs, and other sensory structures belonging to the skeletal motor apparatus of the body. The adequate stimuli for their excitation seem to be mechanical. They contribute to the afferent roots of the cranial fifth and of all the spinal nerves, especially those of the limb regions. Root cells also exist in the eye nerves.

(9) *Visceral and vascular*, mentioned above under dolorous. Normally the sensations they subserve lie little open to introspection, and can, strictly speaking, hardly be considered sensations. Prolonged tensile strain, however, transmutes them into pains. The sacral and thoracic afferent roots and that of the vagus nerve contain these afferent cells, the vagus consisting indeed of little else.

The *efferent root cell* conveys impulses outwards from the nervous system to non-nervous tissues, whose activity is altered by these impulses. The tissues acted on are—(i.) the muscular, both of the skeletal muscles and of viscera, blood vessels, ducts, etc., and (ii.)

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secretory, *e.g.* salivary glands, gastric mucosa, etc. The alteration of activity is in the vast majority of instances an *augmentation;* but in the muscles of the blood-vascular system it is very often *inhibition*, as when the efferent root cells of the *vagus* reduce the activity of the heart, or those of the *corda tympani* relax the musculature of the lingual artery, or those of sacral *n. erigentes* the musculature of the arteries feeding the genital erectile tissue.

THE BULBO-SPINAL AXIS

This central organ into which all these afferent channels lead contains a number of motor mechanisms upon which the nerve impulses brought to it can act. It contains also conducting paths which extend to the brain, and receives from the brain conducting paths leading thence to the spinal motor mechanisms.

Afferent channels from the viscera and the bulbo-spinal mechanisms and efferent channels concerned therewith.— The phenomena of disease, *e.g.* "renal colic," "biliary colic," "referred visceral pains," etc., show clearly that though under ordinary circumstances the reactions of the viscera pass unconsciously, and we cannot perceive them or direct our attention upon them, they can "rise into consciousness," and involuntarily completely absorb attention in virtue of visceral pain. The afferent nerves from the viscera excite visceral reflexes that initiate and control reactions of the visceral musculature, *e.g.* of the bladder, perfectly competently for the execution of many normal visceral functions. Some of these visceral functions employ as adjuncts to their own musculature portions of the skeletal musculature. Thus in micturition the action of the vesical musculature is supplemented by action of the abdominal and pelvic muscles. The afferent nerves from the viscera call into action these adjunct portions of the skeletal musculature. The bulbo-spinal axis contains therefore a number of what may be called "centres," whence the motor and secretory functions of the viscera and their adjunct muscles are initiated and controlled. This regulation of visceral life is one of the great functions of the bulbo-spinal axis. It is one which is comparatively little subjected to the higher (cerebral) centres; these are concerned with the senses that yield perception of the environment rather than of the body: hence the smallness of that part of its waking day which the healthy mind devotes to attention to visceral life.

The afferent channels from the viscera lie in three groups: one in the third and second sacral nerves, coming from the pelvic viscera (bladder, rectum, uterus, and prostate); one in the white rami communicantes of the sympathetic, extending from the second lumbar to the second thoracic inclusive and coming from the abdominal viscera; and

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one in the vagus nerve, coming from the stomach, pancreas, heart, and lungs.

The efferent channels are similarly grouped; they control the whole alimentary canal and its appendages, and the whole vascular system with its tree of contractile tubes.

Deglutition.—Swallowing is in the main a visceral reflex. Volition induces deglutition by placing a bolus against the mucosa of the soft palate and so exciting the afferent fibres of the trigeminus. Then follows co-ordinate discharge of the motor cells of the trigeminus, glossopharyngeal, vago-accessory, and hypoglossal. "Swallowing" consists in the successive contractions of five muscular segments, the mylohyoids, the constrictors of the pharynx, the upper or striated segment of the œsophagus, the middle piece of the œsophagus, and the cardiac segment of the œsophagus. The deglutition centre is not a circumscribed collection of nerve cells, but rather portions of nuclei of origin of the efferent fibres of the above-mentioned nerves. The contraction that sweeps over the œsophagus is of reflex propagation not simply conducted muscularly from one part of the tube to another. A particular patch of the *mucosa* of the soft palate is the most effective reflexigenous area. The total time between the initiation of the discharge in the motor cells of the trigeminus (mylohyoids) and the initiation of the discharge in the motor cells of the vagus to the lowest segment of the œsophagus is about six seconds. A second act of swallowing initiated within six seconds of a previous act inhibits the part of the discharge which has not yet occurred, so that the constriction does not reach the lower end of the œsophagus until six seconds after the second swallow commenced.

Secretory reflexes.—The saliva, the gastric juice, and the pancreatic juice are all secretions which can be reflexly excited, the main afferent channels for the first being the lingual branch of the trigeminus and the glossopharyngeal, that for the two last the gastric branches of the vagus. Little is known of any reflex flow of bile or urine, but the mammary secretion is notoriously influenced by reflex channels, although the secretory nerves of the mammary gland remain unproven by experiment.

Respiration.—The main respiratory centre lies close to the entrance of the vagus fibres into the bulb. It is bilateral, each half presiding over the movements of its own side. But stimulation of one vagus accelerates the discharge from both centres, and section of one vagus renders less frequent the discharge from both. The centre may be considered to consist of an expiratory and an inspiratory division, and with activity of the former slowing of the rhythm with that of the latter quickening of the rhythm occurs. The discharge of impulses from the centre is a rhythmic reply to a constant stimulus, that stimulus being the chemical condition of the blood circulating through

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the bulb itself. The rhythmic discharge of the centre continues after section of all the afferent nerves that can affect respiration. The importance of the composition of the blood for the stimulation of the centre is manifest in the increased activity of it when the blood is deficient in oxygen, contains excess of CO_2, or is loaded with products of muscular metabolism. The periodic activity inherent in the respiratory nerve cells is importantly influenced, and in a practically continuous manner, by centripetal impulses. Section of the vagi slows and deepens respiration. The vagi contain fibres which affect the inspiratory as well as the expiratory centre. Weak stimuli cause expiratory rather than inspiratory effects. Fibres exciting expiration lie especially in the superior laryngeal and the glossopharyngeal nerves. Stimulation of the mucosa of the larynx, and the act of swallowing, especially in its pharyngeal stage, reflexly inhibit the inspiratory movement. Inhalation of irritant gases causes respiratory arrest by the afferent fibres of the trigeminus in the nose, or of the vagus in the larynx and lung. Some gases affect the former, some the latter, others both; some arrest in the inspiratory phase, others in the expiratory. Odours may also arrest. Excitation of the splanchnic nerves usually arrests in expiration, of the sciatic and somatic afferent nerves usually increases the force and frequency of the inspirations, and similarly the cold douche, slapping the skin, etc., increase and accelerate inspiration. External heat is the most potent ordinary means of exciting the inspiratory centre.

Besides the bulbar there are other foci in the brain which are intimately connected with respiration.

Uterine activity.—Parturition has run a normal course in paraplegia. The labour is then unaccompanied by any feeling of pain, but at the height of each of the "pains" of the second stage an indistinct sensation of tightness has enabled the patient to say that the "pain" was present. After complete translesion at the seventh thoracic level lactation has followed upon delivery as usual. Menstruation can continue regularly after translesion of the cord in the thoracic region. The sensory nerves for the uterine body enter the cord by the three last thoracic and the first lumbar roots, and those for the *os* by the second, third, and fourth sacral. This is based upon the situation of the referred pains. The vagina does not give rise to referred pains; its afferent roots are probably the third sacral and the second and fourth.

Penile erection and seminal ejaculation.—These sexual reflexes are prominent in young adults after total transverse spinal lesions in the thoracic and cervical regions. The skin of the genital surfaces and of the front of thigh is a region whence almost any stimulus elicits erection; actual ejaculation is less common. The priapism of paraplegia is in part caused by the paralytic hyperæmia of the erectile tissue. The nervi-erigentes leave the cord by the first three sacral roots.

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Rectum and anus.—In paraplegia the introduction of a compressible bag (Gowers') within the sphincter excites, first, a brief contraction of the sphincter, then a temporary relaxation, and finally a long-lasting contraction. In cases where the lower part of the cord is suffering from severe shock or structural damage the anus may be patulous, and the introduction of a foreign body within it excites merely a temporary contraction. The anal "centre" seems to lie at the third, fourth, and second sacral segments. In the paraplegic dog defæcation can be easily excited by pressure on the pelvic viscera through the abdominal wall, or by irritation of the anus, and with it is usually excited micturition.

Urinary bladder.—Micturition involves two reactions from the visceral musculature, a contraction of the detrusor muscle and a relaxation of the sphincter muscle. Experimental transection above the second lumbar root does not markedly alter the resistance offered by the sphincter to escape of the vesical contents, but transection below that level down to the front of the third sacral segment greatly lowers the resistance. The spinal centre for the tonus of the vesical sphincter extends therefore between those levels; with it is almost certainly combined that for the vesical detrusor. It is probable that the region is divided into an upper, less important (lumbar) portion, and a lower (sacral), more important centre. The motor nerves for the vesical musculature are thus grouped, the weaker issuing by the third and fourth lumbar roots, the stronger by the third and second sacral; and similarly with the afferent nerves. In paraplegic animals micturition is easily excited by pressing lightly on the distended bladder through the anterior abdominal wall.

The paralysing effect of translesion of the cord on the skeletal musculature has no complete counterpart in visceral function. But there is an interference not inconsiderable, though no more marked in the monkey and man than in the rabbit: the "shock" on the skeletal musculature is vastly greater in the former than the latter.

When the viscera are diseased they may disturb consciousness by pains and regions of tenderness in localities supplied by the cutaneous afferent root cells belonging to the same spinal segments as do the visceral afferent root cells. Hence the *referred pains* and areas of tenderness of visceral disease elucidated by Dr. Head are arranged on the body surface not in accord with the distribution of peripheral nerve trunks, but with that of the skin-fields of the spinal ganglia. By observing the position of the area of referred pain and tenderness it is possible, the segmental distribution of the skin-fields of the spinal ganglia on the body surface having been determined, to infer from the segmental spinal position of the pain the viscus exciting it. Working in this way, Dr. Head has furnished the following scheme of the topography of the afferent channels from the viscera:—

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Heart—ventricle—2nd, 3rd, 4th, 5th (and ? 6th) dorsal.
auricle—5th, 6th, 7th, and 8th dorsal.
Lungs—3rd and 4th cervical.
3rd, 4th, 5th, 6th, 7th, 8th, and 9th dorsal.
Stomach—4th cervical.
6th, 7th, 8th, 9th (and ? 10th) dorsal.
Intestines—9th, 10th, 11th, and 12th dorsal.
Lower part of large gut and the rectum—2nd, 3rd, and 4th sacral.
Liver and gall bladder—7th, 8th, 9th, and 10th dorsal.
Kidney and ureter—10th, 11th, 12th dorsal, and 1st lumbar. (? 2nd lumbar).
Bladder—11th, 12th dorsal and 1st lumbar.
2nd, 3rd, and 4th sacral.
Prostate—10th and 11th dorsal.
1st, 2nd, and 3rd sacral.
Testis and Epididymis—10th, 11th, 12th dorsal, and (? 1st lumbar).
Ovary—10th dorsal.
Ovarian appendages—11th, 12th dorsal, and 1st and 2nd lumbar.
Uterus—10th, 11th, 12th dorsal, and 1st (and ? 2nd) lumbar.
(? 1st), 2nd, 3rd, and 4th sacral.

The heart, lungs, stomach, pancreas, and upper part of the small intestine possess afferent channels additional to the above in the *vagi*. In affections of these organs some of the areas of reference are cranial and cervical. These have been especially elucidated by Dr. Head.

As to the path or paths by which centripetal impulses originated by the viscera get access to the sensorium and evoke pain little is known. They may be the same as those of cutaneous pain. Even then it is not clear by what path they ascend. The path soon after its entrance into the cord certainly seems to decussate in part, but whether in greater or in lesser part is a matter of dispute.

Vascular reflexes.—In the bulb, rather headward of the respiratory centre, there is another bilateral centre. Destruction of the brain above this place affects the blood pressure very little; but destruction at or below it lowers the arterial pressure very greatly indeed. This bulbar centre is in constant action, bracing the constrictor mechanism of the blood vessels. It is quickly and powerfully inhibited by the afferent channels coming from the heart in the *depressor* branch of the vagus nerve. *Vid* the spinal cord itself reflex inhibition of the vessels of a local area can be produced, for instance, distension of the erectile tissue reflexly through the lumbar cord. The usual character of a vascular reflex is that dilatation is caused in the region whence rise the centripetal impulses and more or less constriction in the other areas of the circulation; the latter has of course the greater effect on the general arterial pressure, so that an increase of general arterial

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pressure and the local relaxation conspire to ensure increased blood supply to the tissues of the particular region excited.

Muscular reflexes.—The muscles and joints are supplied with sensory end organs and afferent nerves. These are the peripheral apparatus of "muscular sense." When these are destroyed the tonus of the skeletal muscles is impaired in the region corresponding with the damage. By exciting the afferent nerves of muscle either with faradisation or by stretching the muscle or suddenly relaxing it, reflex

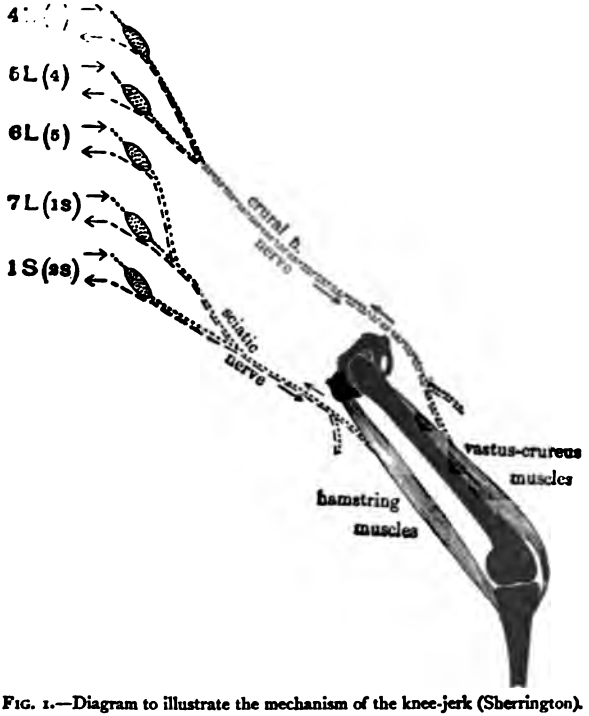

FIG. 1.—Diagram to illustrate the mechanism of the knee-jerk (Sherrington).

movements executed by the local musculature can be excited. Thus flexing the ankle in a paraplegic animal easily excites flexion of the knee and hip of the same side, and extension of hip and knee of the opposite. In such reflexes it is found that the active contraction of the flexors of the knee is accompanied by inhibition of the tonus of the extensors of the same knee. Owing to this inhibition of the tonus of the extensors of the knee the knee-jerk is temporarily abolished or diminished by any reflex that causes contraction of the flexors of the knee.

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The *knee-jerk*, though not itself a reflex, is a delicate index of the reflex spinal tonus. It is a simple spasm of part of the quadriceps extensor muscle, usually elicited by a tap or other brief mechanical stimulus applied to the muscle fibres mediately through the tendon. The contraction is a *direct* reply to a stimulus applied more or less mediately. The reply is obtainable only from muscle fibres possessed of their "neural" tonus. The jerk is more easily obtained when cerebral influence on the spinal cord has been interrupted, or diverted from the knee—for example "reinforcement" by clasping the hands. Transection of the spinal cord above the lumbar enlargement depresses the knee-jerk for a time: in the rabbit for a few minutes only, in the monkey usually for much longer, often for several days, in man for weeks or months. Anæsthetics rapidly abolish the knee-jerk. Depriving the peripheral structures themselves of blood—for example, by application of an Esmarch bandage—abolishes the jerk more slowly. Under abnormal conditions, the muscular reply, even in response to a single tap, is sometimes not a single but a multiple spasm. When the stimulus is prolonged, for example, by depressing the patella (or in calf muscles by depressing the heel), a "clonus" results. The knee-jerk is sometimes spoken of as a "tendon reflex." The shortness of interval between the tap and the beginning of the resultant spasm excludes the possibility of reflex development. The time of the crossed knee-jerk is five times as long (Burkhardt) as of the uncrossed. The crossed knee-jerk may be truly reflex.

In *sleep* the knee-jerk becomes depressed, even to complete abeyance. Extreme bodily fatigue diminishes, and occasionally abolishes the knee-jerk for a while; rest restores it. Fatigue of the extensor muscles of the knee, without general fatigue, has been found to diminish the jerk.

At least four modes of "reinforcing" the knee-jerk are of easy application: (i.) repetition of the tap upon the tendon; (ii.) rubbing of the skin of the limb itself, or still better the use of a cold bath; (iii.) some stimulus through the special senses, such as by a loud clapping of the hands; (iv.) willed movement of the arm. The knee-jerk was found present in every one of 2403 healthy children examined in succession. It is very brisk in young children; ankle clonus and a clonic knee-jerk are said to occur in a large proportion of healthy children. The knee-jerk is said frequently to be absent in old people of normal health. Sternberg, using the devices for reinforcement, found that the knee-jerk is hardly ever really absent in healthy people, although some examined by him were over ninety years of age. Sexual excess tends at first to exaggerate the knee-jerk.

The restraining influence of the cerebrum on the jerk is evidenced by the exaggerated knee-jerk obtained in "decerebrate rigidity." Ziehen has noted the increase of jerk following extirpation of a cortical centre. After decapitation in man the knee-jerk continues obtainable for a minute or more.

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A brief reference may here be made to the *general spinal arrangement of the innervation of the skeletal muscles.* Some muscles are supplied by the motor cells of *one* spinal segment only, such are the intercostales and a few muscles in the limbs, *e.g. tensor fasciæ femoris.* Most muscles are supplied from several contiguous spinal segments. In the trunk and in the proximal portions of the limbs the muscles, although supplied by several spinal roots, exhibit more or less separate territories of their tissue belonging to this and that root. In the smaller and more distal muscles of the limbs the root supply to the individual muscles is not only multiple but the distribution of each root's fibres in each muscle is so commingled that under inspection the whole muscle appears to contract under excitation of each of the nerve roots contributing to its innervation. The motor cells for a single muscle are scattered through a considerable length of cord commingled with the motor cells of the other muscles innervated by that segment. Each filament of the motor root, when stimulated, causes contraction of practically all the muscles which the whole root supplies, so great is the commingling. Hence it comes that no focal lesion of the cord can damage, far less completely paralyse, a single muscle alone; it will tend to weaken a number, but it cannot paralyse one alone. Most of the muscles of the limbs receive nerve fibres from three spinal segments. The afferent nerve fibres of a muscle enter the cord by the afferent roots exactly corresponding with those efferent roots which innervate the muscle.

True deep reflexes.—Although the "jerks" are not reflexes, true reflexes can be elicited by mechanical stimuli applied to tendons, fasciæ, periosteum, bone, etc. A smart tap on an accessible tendon generally evokes a responsive spasm in one or more adjacent muscles. Thus: the inner femoral condyle and the inner malleolus the adductors of the thigh. The sensitivity of bones is well examined by a vibrating tuning-fork.

The spinal path of muscular sense is probably the column of Goll for the lower limb, and for musculature as far forward as the second thoracic segment; for the upper limb it is the median part of the column of Burdach. The path appears in the cord to keep to the same side of the median plane as that on which it entered.

The skin.—Each even small area of the skin contains sense organs attuned to different adequate stimuli subserving four different classes of sensation. Scattered almost everywhere through it are "touch spots," "cold spots," "hot spots," "pain spots." Any given area of the skin seems to possess tactual sensory nerve fibres entering the cord *vid* two spinal ganglia.

As in the musculature, so in the skin, the degree of overlapping of the nerve-root distribution for touch as well as for pain is greater at the distal end of the limb than elsewhere. In the skin of the hand and foot are regions innervated through three consecutive spinal ganglia,

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whereas in most parts of the limb, as in the trunk, the supply of any one area is by two consecutive roots only. The amount of overlap of the right with left across the median line varies in various parts; in the tongue it is slight; on the front of the chest it is considerable. The overlapping is greater for touch nerves than for pain nerves.

The skin reflexes obtainable in a paraplegic animal are much more numerous and energetic than can, as a rule, be elicited in a paraplegic man. Broadly presented by various types of the animal series, one interpretation they will bear seems unmistakable. The movements executed are such as remove sources of irritation from the skin itself and keep it functionally in order. Reflexes are provoked with special ease from certain regions of skin. The most important are the plantar reflex, the drawing up of the testis (cremasteric) of the same side on scratching the skin of the inner part of the thigh, the contraction of muscles in the abdominal wall on scratching the skin over the side of the abdomen, contraction of the gluteus on irritating the skin of the buttock, and the flexion at hip and knee on pricking the skin of the lower limb.

As to the path taken by impulses from tactual, thermal, and dolorific end-organs in ascent to the brain, there is a difference of opinion. Some observers conclude that the path in the main decussates soon after entering the cord; others deny this. It certainly becomes bilateral a short distance above its entrance. The dissociative forms of anæsthesia occurring in disease, *e.g.* syringomyelia, and noted experimentally after certain lesions of the cord, indicate that the spinal paths from "touch spots" and "cold spots" probably lie together and apart from those from "warm spots" and "pain spots," which latter probably run together. The latter seem to enter the gray matter very soon after entering the cord, and then to cross in large part to the opposite lateral column. The two former probably do not decussate so soon or to such a high degree. The view that a lesion breaking through one lateral half of the cord induces anæsthesia on the crossed side below the lesion and hyperæsthesia of the uncrossed is not borne out by observations on semi-section of the cord in animals. Further, the clinical cases where, after cord injury, anæsthesia and hyperæsthesia of the above kind have been observed have not been proved to have been semi-transverse lesions. A transverse lesion of the cord, if it invades the dorsomedian part of the ventrolateral column, usually establishes in addition to some diminution of sensitivity below the lesion a zonal area of hyperalgesia just above the level of the lesion.

In the pons the path for all kinds of cutaneous sensation is found in the median division of the fillet. The upward destination of the fillet is the ventral portion of the optic thalamus and the cortex of the Rolandic area, especially of the post-central convolution. The channels of the muscular sense also lie probably in the fillet.

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As to what centres in the brain receive these various sensory channels, those of muscular sense may lie in part in the cerebellum; lesions of that organ, so far as they cause sensory disturbance at all, interfere with muscular sense, not with cutaneous. The great area of cortex, from islanded patches of which movements of the trunk, and limbs, and face of the crossed side can be elicited, is considered by some to be the chief end-station for the cutaneous senses as well as for the muscular sense. Others regard this Rolandic region as of subsidiary importance to skin sensations, which they connect especially with the mesial aspect of the cortex and the *gyrus fornicatus*. There is experimental evidence, but not of a conclusive kind, to show that when the latter is damaged, little injury being done elsewhere, a condition approaching to cutaneous hemianæsthesia is produced on the crossed side. On the other hand, the destruction of a not too small area of the Rolandic cortex often entails, besides the crossed motor paresis, *e.g.* a brachioplegia, much blunting of sensitivity, especially of localising power, in the skin of the paretic limb. There is considerable impairment of muscular sensitivity in the paretic limb. The sensory paths from the limb seem, therefore, as they ascend, rather to diverge than to converge.

CELL SYSTEMS OF THE SPINAL CORD

Root cells.—The afferent root cells lying in the spinal ganglia thrust each a cellulifugal stem process—an axon—into the cord, which then in the dorsal column bifurcates (Nansen) into an ascending branch and a descending. From these ascending and descending stems collaterals pass horizontally into the gray matter. These collaterals are most numerous in the neighbourhood of the entrance of the dorsal root. Some of the collaterals pass direct across to the motor-horn cells; these are the so-called "reflex collaterals"; others pass to the cells of Clarke's column, which cells give axons to the superior vermis of the cerebellum ascending the peripheral of the lateral column as the cerebellar tract; others finally end in Goll's and Burdach's nuclei in the bulb.

Of the efferent (motor) root cells, two classes can be distinguished —(1) To the skeletal muscles, and (2) to the unstriped muscles of the skin (*erectores pilorum*), blood vessels and viscera, and to secretory cells. In the spinal cord both lie almost exactly at the same level as the place of exit ("surface origin") of the nerve fibres to which they give rise. The second class are probably quite small cells; they give origin to small fibres of 2.5μ diameter and less; and they are absent from certain spinal segments, *e.g.* from the three lowest lumbar and two highest sacral roots, and from the cervical and first thoracic. In the bulb they form the *nucleus ambiguus*, and in the thoracic region they very probably are the cells of the lateral horn.

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Mediate cells.—All other than the root cells afferent and efferent belong to cell systems which are *mediate*, that is, link one root cell to another. The spinal mediate cell systems are classed as the intrinsic or purely spinal, and the extrinsic or spino-encephalic and encephalo-spinal.

Intrinsic mediate systems.—These form a large portion of the fibres of the white matter. Some ascend; some descend. Some of the former—though only a small fraction of them—cross from one lateral half of the cord in the ventral commissure. Of the latter most do not decussate but lie in the ventro-lateral column, and partly in the dorsal columns, forming the septo-marginal tract, the comma tract of Schultze, and similar small tracts.

Extrinsic mediate systems—(1) *Spino-cerebellar.*—These form a more or less continuous zone of fibres, for the most part large, along the whole periphery of the ventro-lateral column, except in the deepest part of the ventral median fissure. They ascend to *superior vermis cerebelli*, those in the dorsal part of the lateral column passing by the inferior peduncle (*corp. restiforme*), those in the ventral by the superior peduncle (*brach. conjunctivum*). Their origin is in part from the cells of Clarke's column, which lie chiefly in the spinal segments from the second thoracic to second lumbar inclusive.

(2) *Spino-quadrigeminal.*—There is some evidence that certain spinal cells, the position of which is not clearly ascertained, send axons to the quadrigeminate bodies. These probably co-ordinate eye with limb movements, or with trunk movements. The fibres lie in the ventral root zone of the cord, and ascend the bulb in close proximity to the ventral portion of the cerebellar tract.

(3) *Encephalo-spinal systems.*—Cells of the reticular formation of the bulb and pons, and also of Deiters' nucleus—the end-station of the vestibular nerve—give off axons that pass down into the cord and run along its whole length in the ventro-lateral columns. Some of these cross the median plane, in the bulb, and some remain uncrossed. A cell system, with its cell bodies in the "red nucleus" (mesencephalon), also sends fibres into the ventro-lateral spinal column, chiefly of the crossed side. There is evidence further of fibres which pass down the cord in the lip of the ventral fissure from anterior corpus quadrigeminum—these fibres for the most part cross the median plane near their origin.

But the best known of the encephalo-spinal systems is the *pyramidal.* The fibres of this are axons from certain cells in the Rolandic cortex. They descend through the internal capsule, *pes pedunculi*, pedal longitudinal fibres of the pons, and the pyramid of the bulb, of which last they compose the ventral four-fifths. The "decussation of the pyramids" is the crossing in bulk of the bundles of this system at the caudal end of the bulb. The vast majority of its fibres cross into the dorsal part of the lateral column of the cord. This crossed bundle of the tract extends the whole length of the cord, but diminishes

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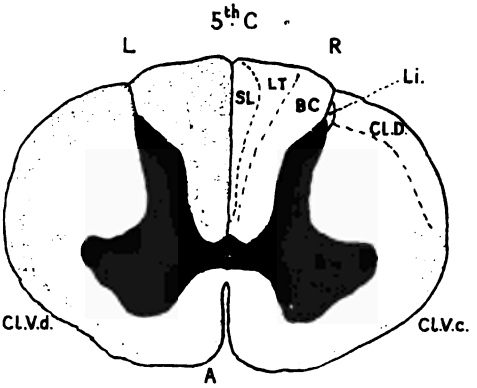

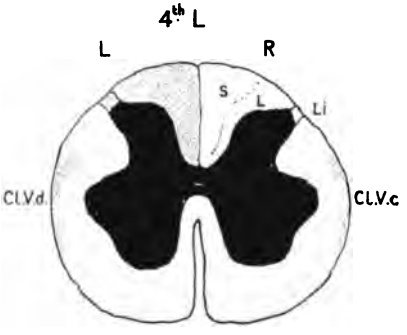

Ascending Tracts.

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PLATE I

Ascending Tracts

L=left side of cord; *R*=right side of cord.

Diagrams of the cord in cross section at the fifth cervical and fourth lumbar levels, to show the positions of ascending tracts of fibres. *S*, fibres of the sacral afferent nerve roots; *SL*, fibres of the afferent roots of the sacro-lumbar nerves; *LT*, of the lumbar and thoracic roots; *BC*, of the brachial and cervical nerves; *L*, of the lumbar nerves; *Li*, small ascending fibres, tract of Lissauer; *Cl. D.*, dorsal cerebellar tract reaching cerebellum by the restiform body; *Cl. V. d.*, ventral cerebellar tract reaching cerebellum by superior peduncle, uncrossed portion; *Cl. V. c.*, crossed portion; *A*, ascending fibres of unknown destination (Laslett and Warrington).

The comma-shaped area left less deeply stippled in the dorsal column marks the position of the descending comma tract (Schultze).

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PLATE II

DESCENDING TRACTS

L=left side of cord; *R*=right side of cord.

CORTICO-SPINAL TRACTS

Diagram of the spinal cord in cross section at the fifth cervical and fourth lumbar levels, to show the position of the tracts of fibres descending from the cerebral cortex of one hemisphere, *i.e.* the left. *Py. V.*, the ventral bundle of the pyramidal tract; *Py. D. c.*, the crossed bundle of the dorsal portion of the pyramidal tract; *Py. D. d.*, the uncrossed bundle of the dorsal portion of the pyramidal tract.

ENCEPHALO-SPINAL TRACTS OTHER THAN CORTICAL

Diagram of the cord in cross section at the fifth cervical and fourth lumbar levels respectively, to show the positions of the tracts of fibres descending from parts of the brain behind the hemispheres. *Ms. B. sp. L. d.*, tract descending from the region of the corpora quadrigemina and red nucleus and lateral part of the bulb into the lateral column of the same side, *i.e.* the uncrossed portion of the lateral mesencephalo-bulbo-spinal tract; *Ms. B. sp. L. c.*, crossed portion of the same tract; *Ms. B. sp. V. d.*, similar tract descending in the ventral column (contributing to the sulco-marginal tract (*Su. m.*) of Marie), partly crossed, partly uncrossed.

DESCENDING LONG INTRA-SPINAL TRACTS

Diagram of cross section of cord at the fourth lumbar level, to show position of the longer spinal tracts arising in the cervical and thoracic regions of the cord itself and descending into the sacro-lumbar. *Sp. l.*, lateral portion of the long spinal tract; *Sp. V.*, ventral portion of same, contributing to the sulco-marginal tract (*Su. m.*) of Marie; *St. m.*, the septo-marginal tract of Bruce; *Cc*, cornu commissural bundle from thoracic region. This diagram, if combined with the corresponding ones above, gives the degeneration below translesion of the cord itself.

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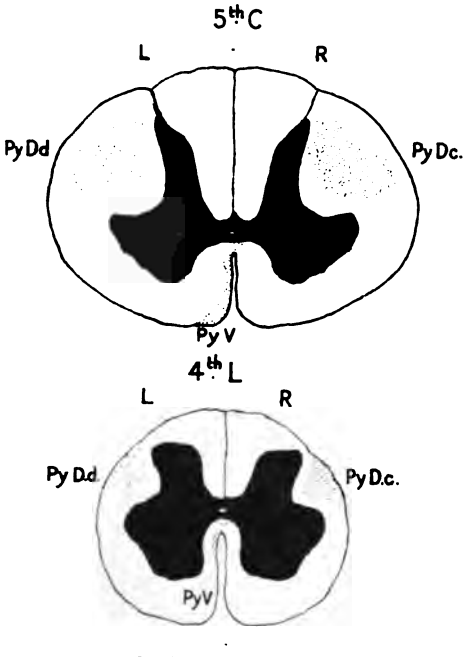
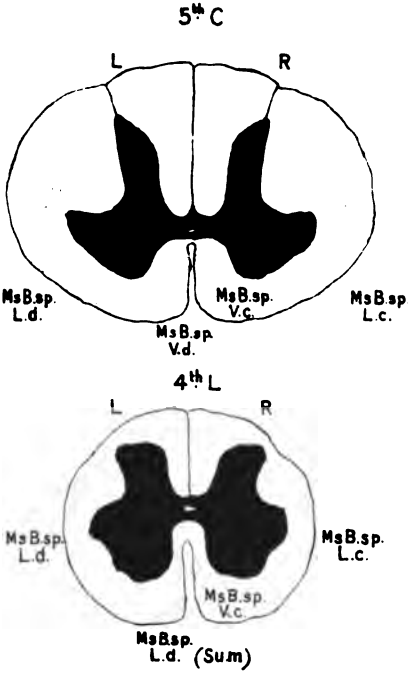
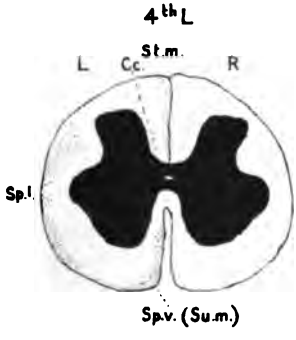


Cortico-spinal Tracts.



Encephalo-spinal Tracts, other than Cortical.



Descending long Intra-spinal Tracts.

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as it descends, constituent fibres stopping short at various levels. Its progressive decrease is far from regular, at some regions many fibres ending, at others comparatively few. Of the minor component of the tract which does not decussate at the bulb there are two divisions—one that ultimately crosses, continuing in fact the bulbar decussation, and extending it for a long distance down the cord, contributing to the white commissure of the cord and hugging the side of the ventral fissure, even into the sacral region. This is Turck's bundle, or the ventral pyramidal bundle. The other division is a truly uncrossed portion that at the level at which the main crossed portion slips over to the opposite lateral column deviates from the median plane into the lateral column of its own side. This uncrossed division also extends throughout the length of the cord.

The pyramidal system is, as far as known, existent only in mammals. It is not the only nerve-path connecting the "motor" centres of the cortex cerebri with those of the spinal cord. After complete destruction of the pyramids excitation of the cortex cerebri still evokes movements of the limbs, etc. In man it is relatively far more voluminous than in any other type. It is the latest of the spinal systems to complete its development, as shown by the late date of myelinisation of its fibres; it is also the slowest to show signs of Wallerian degeneration, in result of traumatic interruption.

In its spinal course the pyramidal system of fibres lies nowhere actually abutting on the ventral gray horn. From the fibres of the lateral column, collaterals pass into the gray matter; some of these are probably from the pyramidal fibres. Direct synapsis between the pyramidal axon and the ventral cornual root cell is generally accredited, though rejected by some.

When the above-mentioned distribution of the cell systems is taken in conjunction with the law of cell life, that a part of a cell not containing the cell nucleus cannot maintain its vitality, it is easy to explain the spinal degenerations which follow traumata of the cord and brain. These degenerations are termed *ascending* and *descending*, according as they lie headward of or below the site of lesion.

THE BULB OR REGION OF THE MEDULLA OBLONGATA (MYELENCEPHALON)

The mammal, after removal of the encephalon in front of the bulb, still breathes with regularity and possesses good circulatory adjustments.

Ablation of the bulb itself annuls the respiratory movements, depresses arterial tension, and destroys a number of vasomotor reflexes that were previously possible. The cardio-inhibitory centre connected with the vagus and accessory nerve is also destroyed; the reactions of the sensory nerves of the body upon the heart beat are lost.

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The nervous "centre" for ingestion of food lies in the floor of the fourth ventricle. It is subdivisible into "centres" of deglutition, phonation, vomiting, coughing, hiccough, etc. The reflex centres of movement of the œsophagus and of gastric and pancreatic secretion lie in the caudal part of the bulb in nexus with the vagus and its accessory nerve, so that myelencephalic influence goes far aborally along the alimentary canal. In the bulb inhibition enters very obviously into "co-ordination." Each subdivision of it is depressible by "inhibitory" fibres from some afferent nerve or another: the respiratory portion by fibres in the superior laryngeal nerve; the deglutition portion by fibres in the superior laryngeal and partly in the inferior laryngeal; the secretory portion by fibres in the afferent branches of the gastric and thoracic vagus, and so on.

Widespread convulsions of the skeletal musculature are produced by chemical stimulation of the bulb. A drop of 10% sodium chloride solution or a crystal of kreatin placed on the floor of the fourth ventricle of the frog produces after a minute or so a cry which ushers in a seizure, during which the fore limbs are crossed under the sternum, the hind limbs at first flexed at hip and extended at knee and ankle until the animal rolls backward. Then extension at hip follows. The muscular contraction, at first clonic, later becomes tonic. The whole seizure lasts about one minute. The seizure is less intense if the cerebral hemispheres have previously been removed. Convulsions cannot be similarly excited from the spinal cord itself.

Concerning the bulb as a conductor the following résumé must suffice. There descends into the cord from the crossed half of the mesencephalon a path that probably co-ordinates by single linkage movements of the body with stimuli impinging on the retina. From the end-stations of the vestibular nerve descends into the cord an uncrossed path that probably relates the body movements with the sensations originated in the semicircular canals. Paths also, both uncrossed and crossed, descend into the cord from the red nucleus, and the large diffuse nuclei of the reticular formation of both middle and hind brain, and the functions of these are even more conjectural. Finally, there is in mammals a single link path descending from the cerebral hemisphere into and through the bulb to reach the motor root-cell system.

The excitation of each pyramid evokes movements chiefly in the contra-lateral skeletal muscles especially of the limbs, but to some extent also in the homonymous. Complete severance of both the pyramids does not, however, produce, even in so high a type as the dog, any obvious permanent paralysis of movement, nor does it prevent in the dog excitation of the Rolandic cortex producing movement both on the crossed or uncrossed limbs. Pyramidal fibres have been traced into the hypoglossal nucleus, but not into the other motor nuclei of this region, and only into the contra-lateral hypoglossal.

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Of the paths ascending through the bulb from the spinal cord the chief are the continuation from the dorsal columns of root fibres to pass by a second link from the dorsal column nuclei to the fillet of the crossed side, the spino-cerebellar one chiefly uncrossed from the cord and entering the inferior peduncle of the cerebellum, the other chiefly crossed and entering with the superior peduncle of the cerebellum. With this latter pass probably spino-mesencephalic and spino-thalamic paths towards end-stations, as their names imply, in front of the bulb itself. The path from the dorsal column nuclei, *vid* the crossed fillet, extends to the diencephalon (optic thalamus) and in part to the cerebral cortex, especially its parietal region. Of the function of the inferior olive little is known; its connection with the paths from the vestibular nerve is suggestive.

THE REGION OF THE PONS VAROLII AND CEREBELLUM (THE METENCEPHALON)

The floor of the metencephalon contains the median fillet, with its constituents from the dorsal column nuclei. Near it the lateral fillet contains ascending paths from the end nuclei of the auditory nerves chiefly contra-lateral. The superior olive is, to judge from its connections, probably of auditory function. In the more lateral region ascend paths from the ventro-lateral column of the spinal cord, chiefly from mediate cells in the opposite half of the gray matter. They are on their way to the roof of the metencephalon (cerebellum) and the mesencephalon. Transection of the dorsal portion of the metencephalic floor to one side of the median sagittal plane produces (monkey) complete anæsthesia (including analgesia) of the contra-lateral half of the body without any paralysis. Of the paths connected with the cerebellum most notable is the ascending of the inferior peduncle, coming from the lateral column of the cord and Clarke's column of cells (uncrossed mediate cells of the cord). There is also a path conducting upward from this region into the crossed corpus striatum. There descend into the pons three main paths from the cerebral cortex: one from the frontal cortex which ends in the gray matter of the pons; a second from the temporal cortex which ends in the gray matter of the pons; and a third, the pyramidal, which only in small part terminates in the pons, in motor nuclei there. Of these the fronto-pontine is the most lateral, the temporo-pontine the most median. Of the fibres of the pyramidal tract which end in the pons, some have been traced to the crossed nuclei of the motor fifth and seventh, and some also to the homonymous nuclei. From the "reticular formation" of the pons a descending path passes into the ventro-lateral columns of the cord, partly decussating in its descent. There also pass through the reticular formation of the pons paths descending from the mesencephalon (red nucleus and anterior corpus

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quadrigeminum) to the ventro-lateral column of the spinal cord. The posterior longitudinal bundles form two more paths, chiefly descending. They mediate between the motor cell groups of the ocular and neck muscles.

THE CEREBELLUM.—Atrophy of one lateral lobe of the cerebellum is associated with that of the crossed cerebral hemisphere. The superior vermis receives the endings of the spino-cerebellar tracts that ascend in the margin of the ventro-lateral columns of the spinal cord, and are of spinal origin, chiefly uncrossed. Faradisation of parts of the superior vermis gives conjugate movements of the eyes. Ablation of half the cerebellum produces characteristic symptoms. Supposing the half extirpated to be the right, the animal lies on that side with neck and trunk curved with concavity to the right, but with torsion and deviation of the head toward the left. There is often conjugate deviation of the eyeballs to the left. The creature cannot stand, but falls to the right. A few days bring some recovery of ability to stand. The right limbs are abducted. There is some extensor rigidity in both limbs, and especially some paresis in the hind limbs. Tremor disturbs every effort, especially of the neck and trunk. The gait is reeling, with curves "festooning" to the right. The knee-jerk is often more brisk right than left. The animal tends in walking to deviate to the right, in swimming to the left. Incoordination is not obviously increased by bandaging the eyes. Sight, hearing, and cutaneous sensation seem sound. Gradual improvement ensues, but disappearance of the symptoms is never complete, thus the animal in shaking itself after a swim is apt to shake itself off its balance, falling to the right.

If the whole cerebellum be removed the disturbance seems at first less severe than after unilateral lesion. There is opisthotonos, retraction of the neck, bilateral extension and abduction of the limbs, especially the fore limbs. Every attempt to move induces tremor. The hind limbs are paretic. Recovery is much slower than after unilateral lesion. Luciani preserved one of his dogs for two years; the animal could swim, run, jump, and feed itself, but tremor interfered with all its "intentional" movements. Ablation of the middle lobe alone causes opisthotonos, abduction and extensor rigidity of the limbs, but the symptoms subside rather rapidly.

Occasionally there come to light congenital defects amounting to absence of one cerebellar hemisphere. In some this huge defect has occasioned practically no symptoms. Movement has been neither uncertain or tremulous. In one case, the right being the agenesic hemisphere, the patient had "a habit" of keeping the head turned somewhat toward the left. Of unsteadiness of gait, of weakness of limbs, nystagmus, peculiarity of speech, vertigo or defective sensation or intelligence, it is certain there was no trace. The knee-jerks were normal and equal right and left. In rarer cases there has been arrest of growth

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of the whole cerebellum. Not a hundredth part of the organ has remained, and yet there was ability to stand, walk, handle delicate objects, in fact, a fairly normal volitional motility was preserved. Noteworthy in such cases has been absence of detectible impairment of cutaneous sensitivity. The remarkably negative character of even extreme cases of cerebellar defect is paralleled by the frequency with which cerebellar lesions of even large size but non-irritative type await unsuspected a post-mortem revelation. When the lesion is sudden and fulminant, the symptoms are those which, in their complexity, have been indicated above. They have yielded to analysis by Luciani the essential defects of ataxia, parasthenia, or weakness of certain muscle groups, and paratonia or deficiency of tonus in the muscle groups (see page 131).

THE REGION OF THE CORPORA QUADRIGEMINA (THE MESENCEPHALON)

The nervous masses of the "roof" of this region of the nervous axis form, in mammals, the corpora quadrigemina. In the dog it is possible to elicit different co-ordinate eye movements from different points of the anterior corpora: from the sulcus between them return of the divergent bulbs to parallelism without any change in the pupils; from a posterior point elevat.on of the eyes with dilatation of both pupils; further back still convergence with contraction of both pupils. After sagittal section in the median plane the excitation affects only the homonymous eyeball. In all the conjugate reactions the contraction of one muscle is accompanied by relaxation of the tonus of its antagonist. But the eyeball movements elicited from the corpora quadrigemina are not always conjugate; sometimes one movement only is elicitable from the whole of both the anterior corpora. In the monkey the reactions are similar to those of the dog, with the addition of wide opening of the palpebral fissures, elevation of the eyebrows, and turning of the whole head in the direction of the gaze, *i.e.* away from the side of excitation. Excitation of the posterior corpora produces vocalisation, the sounds varying between a short bark and all varieties of cries. I have found vocalisation elicitable reflexly, *e.g.* from skin, in the monkey after transection close behind the anterior corpora; transection behind the posterior corpora seems to abolish it. The cry is a prolonged cry, and in all the reflexes obtained a deliberate slowness is notable and the attitudes assumed are long maintained. The tonic component of the epileptic discharge into the muscles is not cortical but subcortical. The "cataleptoid reflexes" elicitable after transection at the mesencephalon recall this. After complete transection the condition termed *decerebrate rigidity* supervenes. In this status the respiratory movements proceed regularly and the pulse remains quiet and full, but certain groups of muscles become rigid—the elbows and knees becoming

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stiffly extended and the neck retracted. Section of the afferent roots of the arm prior or subsequent to the transection of the mesencephalon prevents or abolishes the brachial rigidity.

In "*decerebrate rigidity*" reflexes can be elicited which actuate the neck and trunk and the four limbs as a whole. The combined movement treats the pair of fore limbs in such a manner that at the same time the one is flexed and protracted, the other is extended and retarded, and similarly when the one hind limb is flexed at hip and knee its fellow is extended at those joints.

Inhibition is exemplified in the execution of these combined limb reflexes. Thus, reflex inhibition of the left *triceps brachii* with contraction of the flexors of the elbow, and inhibition of the right *extensor triceps cruris* with contraction of the hamstring muscles.

Paths that ascend the mesencephalon and in part end in it, lie in the fillet, and these partly come from the ventro-lateral column of the spinal cord and from the end-nuclei of the auditory nerve. A large path from the interior of the lateral lobe of the cerebellum crosses the mid-line to enter and in part end in the "red nucleus" of the tegmental region. Severance of the tegmental region and fillet on one side of the mid-line produces anæsthesia and analgesia of the skin of the contra-lateral half of the body; the analgesia, at first total in degree, gradually subsides to partial. The posterior longitudinal bundles running up and down the region seem mainly commissural and associational between the ocular nuclei and those of the neck muscles.

Main descending paths of the region are the mesencephalo-spinal, a crossed path traceable from the anterior and posterior corpora quadrigemina into the ventral part of the opposite ventro-lateral spinal column. This may be a projection system belonging to vision. Then there are the cerebral descending paths, fronto-pontine, temporo-pontine, and pyramidal. These in their descent give fibres to the substantia nigra, and from the pyramidal pass fibres to the oculomotorius and trochlearis nuclei, some to the crossed and some to the homonymous. Efferent fibres pass from this region into the retina by way of the optic tracts and nerves. Conversely from the ganglion-cell layer of the retina a path enters the crossed optic lobe, and in mammals partly into the uncrossed. The ratio of the uncrossed to the crossed is highest in animals in which the visual axes are parallel, *e.g.* man.

Other paths are to the corpora striata, to the homonymous half of the cerebellum, to the motor nuclei of the bulb, chiefly of the crossed side. The mesencephalon also receives a path from the corpus striatum.

THE REGION OF THE OPTIC THALAMUS (DIENCEPHALON)

This region includes as its main divisions, study of which in regard to function has been prosecuted, the optic thalamus, corpus subthala-

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mium, and lateral geniculate body. The region sends few if any fibres in a caudal direction; on the other hand, it is an abundant source of fibres to the telencephalon, both corpus striatum and cortex. It is connected with the Rolandic region of the cortex by a system of fibres that, among cortical fibres, become medullate somewhat early, and by later developing systems with (1) the gyrus hippocampus, cingulum, and gyrus fornicatus; and (2) with the frontal convolutions. It is also connected with the cortex of the insular region, and sends an especially numerous fibre system to the occipital and temporal cortex, including pre-eminently that of the calcarine and angular gyri. No region of the cortex of the hemisphere is without nerve fibres from the optic thalamus.

The loss of an eye induces shrinkage of the contra-lateral optic thalamus. Only in Primates is the pulvinar of large proportions; in most Mammalia it is quite small in size. All goes to indicate the growing importance of the cortex for vision as the sense evolves. Of the four masses of gray matter into which, in mammals, the thalamus is broadly divisible, it is the hindmost (pulvinar) that is chiefly in receipt of fibres from the optic "nerve," and from the pulvinar pass fibres to the occipital and to the angular regions of the cortex. The lateral geniculate body also sends fibres to the occipital cortex.

In the lateral gray mass of the thalamus end many of the fibres of the fillet. They enter especially the ventral and posterior part of the lateral mass, and lie around the median centre of Luys. And it is from the lateral gray mass of the thalamus that corticipetal fibres pass to the parietal and falciform (mesial) regions of the cortex.

Lesion of one thalamus produces visual defect, and dilatation of the contra-lateral pupil. In Dr. Hughlings Jackson's case of softening of the posterior part of one thalamus, the symptoms observed were diminution of tactual sense on the crossed side of the body, impairment of common sensitivity in the left nostril, slight impairment of taste on the left side of the tongue, and of hearing in the left ear, and loss of the left half of the field of vision of each eye. Experimental work substantiates this. The defect of vision for the contra-lateral retina seems, at first at any rate, more than a hemianopsia, *i.e.* a hemianopsia plus amblyopia of the whole field.

THE CEREBRAL CORTEX

The arrangement of the nerve cells in the cerebrum is such that the perikarya (cell bodies) and dendrons lie next the surface, forming the gray matter of the cortex, while the axons, which are almost all of them myelinated nerve fibres, pass from and into the gray matter by its deep surface. As the nerve fibres, being narrower, occupy less transverse area than the perikarya with their dendrons, the sectional area of underlying white matter sufficient for the corresponding area of gray matter is

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relatively small. Hence, where the cortex is extensive it is found to be folded, and a relative increase in the gray cortex seems thus obtained. Since, after the fifth month of fœtal life the number of cells in the central nervous system is not increased, the only remaining means of increase in size is by growth in individual bulk of the cells already existent. Thus if the average size of the motor nerve cells in the ventral horns of the spinal cord of a four-week fœtus be taken as 1, the average

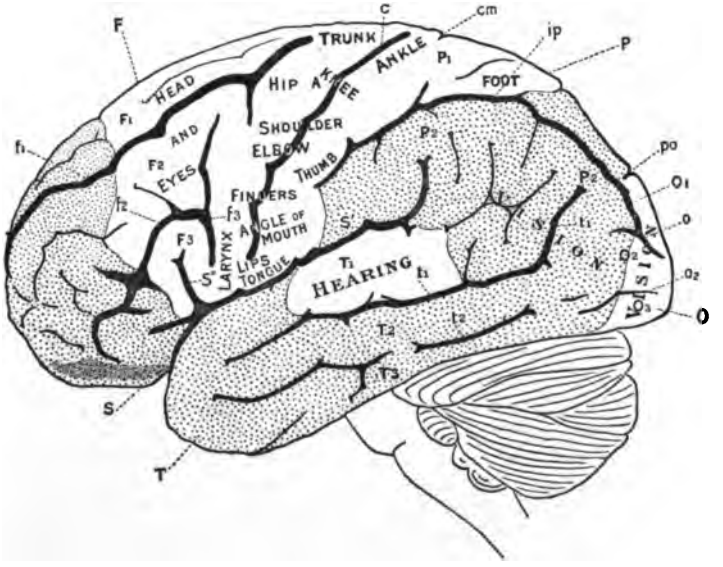

FIG. 2.—The external surface of the left cerebral hemisphere, showing the motor and sensory areas. The shaded portions correspond to Flechsig's "association" centres. (Modified from Ecker and Flechsig, by W. A. Turner.) The lettering indicates the lobes, gyri, and sulci. *F*, frontal lobe; F_1, F_2, F_3, the first, second, and third frontal gyri; f_1, f_2, superior and inferior frontal sulci; *T*, temporal lobe; T_1, T_2, T_3, the first, second, and third temporal gyri; t_1, t_2, the superior and inferior temporal sulci; *O*, occipital lobe; O_1, O_2, O_3, the first, second, and third occipital convolutions; *P*, parietal lobe; P_1, the superior parietal lobule; P_2, the inferior parietal lobule; *S*, the fissure of Sylvius; *S'*, posterior limb; *S''*, ascending limb; *po*, parieto-occipital fissure; *ip*, the intra-parietal fissure; *C*, the fissure of Rolando. Motor areas are marked by small, sensory areas by large capital letters.

size at birth is 124, and in the adult is 160. The cortex of the hemisphere is, in the adult, twice as thick as it is in the eighth month of intrauterine life. The growth of individual nervous elements in the cortex must go on long into adult life, for the number of fibres in its layers has been shown to be distinctly greater at middle life than in early adult life. On the other hand, the number of fibres appears to be less at the age of eighty than at that of thirty-five.

Conducting channels from all the sense organs reach the hemi-

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sphere. Transection through one lateral tegmental region close behind the cerebrum causes hemianæsthesia of the crossed half of the body. If not only the tegmental but the pedal region of the cerebral peduncle be severed, to the hemianæsthia there is added hemiplegia. The paths from the various sets of sense organs diverge to different regions of cortex. Hence there are visual, auditory, tactual, gustatory, and muscular sense regions. The motor apparatus of the body lies to a variable extent at disposal of these various sets of end-organs. This and that portion of the motor apparatus is more or less especially

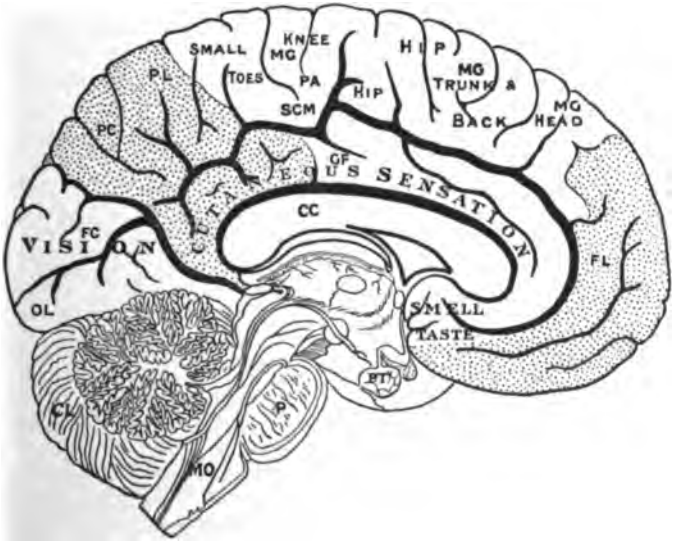

FIG. 3.—The mesial surface of the left cerebral hemisphere, showing the motor and sensory areas, and the "association" centres of Flechsig (shaded). (Modified as Fig. 2 by W. A. Turner.) *FL*, frontal lobe; *GF*, gyrus fornicatus; *MG*, marginal gyrus; *CC*, corpus callosum; *PL*, parietal lobe; *CL*, cerebellum; *PC*, precuneus; *MO*, medulla oblongata; *FC*, cuneus; *P*, pons; *OL*, occipital lobe; *SCM*, sulcus calloso-marginalis.

related to this or that particular species of sense, for instance, the musculature moving the eyeball especially to the retina. Hence on exciting the various sense regions of the cortex certain movements are provoked characteristic for the sense and the region. Investigation, by Hitzig, Ferrier, Munk, Schäfer, Luciani, and many others, has shown the cortex to contain a number of "excitable areas" which, by the distinct motor reactions they severally yield, indicate an almost minute spatial partition of function.

The *genu* of the Rolandic fissure when present in man seems to mark the limit of the facial area below from the brachial above, and lies usually somewhat above halfway between the Sylvian and great

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longitudinal fissures. The division between the arm and leg areas lies about opposite the superior frontal sulcus.

From all this region pass, as has been demonstrated by Wallerian degeneration, fibres of the pyramidal tract to the motor nuclei of the cranial and spinal nerves. This degeneration is, though mainly crossed, not entirely so. Comformably with that fact the movements elicited, although mainly crossed, are in a number of instances bilateral, *e.g.* the opening of the eyelids.

The movements referred to are the first movements evoked on exciting the respective cortical areas. The reactions experimentally observed are on the whole deservedly described as constant, and when such and such a piece of cortical surface is spoken of as "the thumb-centre," what is meant is that when that area is faradised a movement of the thumb is usually the first movement to be elicited, and if the stimulus be weak and not too long continued the thumb movement will be the only movement obtained. The movement of the thumb is not always the same; it is often flexion with adduction, often extension with abduction. What conditions determine whether it be this or that are unknown. The area of cortex whence movement of the thumb is elicitable as a primary reaction is larger than that whence movement at shoulder is similarly obtained. There is no direct relation between the extent of a cortical area and the mass of muscles which it controls. The mass of muscles in the leg is five times greater than that in the arm and this many times greater than that in the face and head; yet for the last the cortical area is greatest. The movement elicited from the cortex tends to outlast the application of the stimulus by an appreciable time; especially is this so when the stimulus is prolonged. As the stimulus is prolonged the movement induced has other movements added to it. Thus to flexion of the thumb may be added flexion of the other digits, then movement at the wrist, flexion at elbow, protraction of shoulder, and even movements of the face and lower limb. This resembles the "march" (Hughlings Jackson) of convulsion in a focal epilepsy. In fact, by prolonging the stimulus applied to the cortex an epileptic attack can be induced, ushered in by the particular movement characteristic for the area stimulated. The kind of muscular contraction elicited from the cortex is tonic at outset but tends to become clonic. The clonic stage of the epileptiform seizure is after all only an exaggeration of the form of contraction which each muscle exhibits under experimental faradisation of the cortex. Under absinthe poisoning each application of the electrodes to the cortex evokes an epileptiform seizure. If the cortex be removed the epileptiform seizures cease, although movements are still evocable from the underlying white matter of the corona radiata; an epileptiform convulsion, when initiated, may even be cut short by rapid extirpation of the cortex of the region involved.

With the movements about the larger joints it is usual to find

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areas of cortex which preside respectively over the antagonistic movements at the joint, thus separate areas for extension and for flexion at elbow. The antagonistic movements about the more proximal joints of the limb are segmentally more separate one from another in the spinal cord than are the antagonistic movements at the distal joints. That *movements*, not *muscles*, are represented in the cortex can be shown very clearly in the case of the joints with separate centres for antagonistic movements, because it is found that both sets of the muscles of the joint are influenced from each of the cortical centres, but from one of the centres, if the joint be for instance elbow, the triceps is made to contract, while the biceps and other flexors are made to relax, while in the other centre the effect is the converse. The movement initiated by faradisation of one of these cortical centres can be, when in process, cut short by faradisation of the antagonistic centre—sometimes this inhibition is induced very readily.

When the "march" or sequence of movements occurring in the limb is examined it is found to vary for different centres of the cortex. Sequences frequently observed experimentally are the following: (1) adduction and retraction of shoulder, then flexion of elbow, then extension of fingers and wrist; (2) flexion and opposition of thumb, then flexion of fingers, then extension of wrist, then flexion of elbow, then adduction of shoulder; (3) great toe, then extension of digits, then flexion of ankle, then of hip, then of knee; (4) flexion of hip, then of knee, then of ankle, then of toes.

The intensities of current required to excite the cortex in man are much greater than in animals. Perhaps this is because the excitable elements in the thick cortex of the human brain lie more deeply than in less developed hemispheres. The period of latent excitation is, for cortical excitation, distinctly greater than for excitation of the *corona radiata*. Faradisation of the cortex does, therefore, actually excite constituents of the cortex and not the fibres of the underlying white matter. The cortex even of the Rolandic region itself is not a continuous field of excitable surface. It is cut up into islets of excitable surface surrounded by inexcitable. These non-excitable zones surrounding the excitable centres are later in development and their fibres become medullate later, and do not contribute to the great projection system of the pyramidal tract. The "projection" or excitable centres are surrounded in the highest types of brain by extensive "associational" areas correlating with the neighbouring projection areas. The projecting systems emanating from the cortical territories pass through the internal capsule. In the cross section of this comparatively limited structure there lie therefore bundles of fibres which, when faradised, give motor reactions resembling those yielded by the cortical centres above. Very limited lesions of the capsule suffice to produce marked paralysis. As these are traced

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in cross sections successively more and more basal they come to lie more and more behind the knee of the capsule, the post-genual limb of the capsule becoming longer and longer.

This Rolandic region, containing areas for the face and arm and leg,

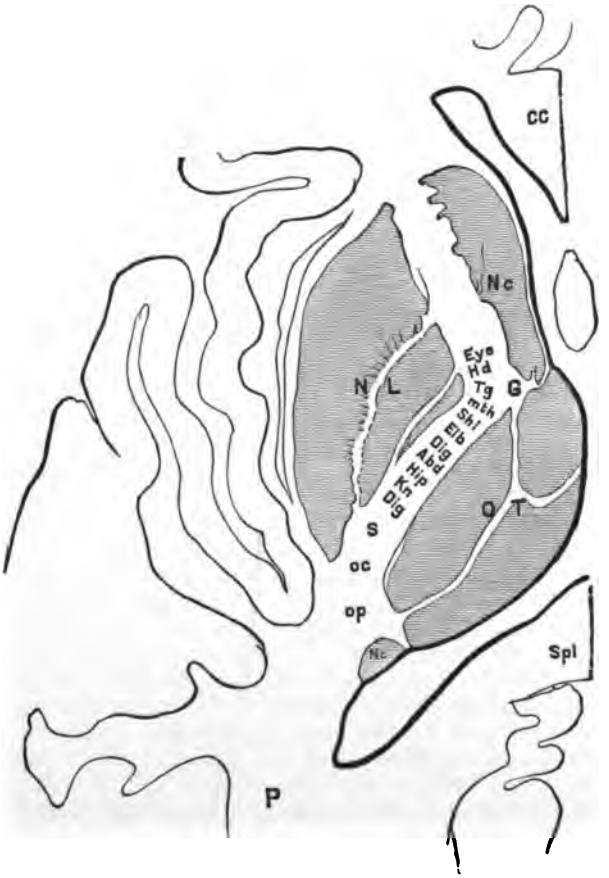

FIG. 4.—Horizontal section of brain to show the relative position of the several constituents of the pyramidal tract in the internal capsule. *G*, knee of internal capsule; *NL*, lenticular nucleus; *Nc*, caudate nucleus; *OT*, optic thalamus. (From Foster's *Physiology*, part iii.)

and to a smaller extent the trunk, is called by some the region of bodily sense or *somæsthesia*. That its function is intimately connected with afferent channels is clear from various circumstances, among others the following :—

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(1) Fibres from the fillet have been traced up into the cortex of this region, and the fillet is a collection of sensory paths.

(2) In cases in which in man this region has been faradised, sensations in the part moved as well as the appropriate movement have been occasioned by the stimulation.

(3) Ablation of the areas of this region, if extensive, in addition to paresis, are accompanied by a disturbance of skin sensation, particularly in the power to localise stimuli (Westphal, Déjerine).

(4) If the afferent spinal roots belonging to a limb be severed, the willed movements employing that limb become ataxic, and movements, *e.g.* grasp by the hand, are lost, apparently permanently. On exciting the appropriate area of the cortex, however, the grasp movement is elicited, although it may have been in abeyance many weeks.

(5) In epilepsy the seizures starting from centres in the Rolandic cortex are often preceded by "aura" localised in the hand, leg, or other part corresponding with the focus of the epilepsy.

(6) There is reason to think that the muscular sense of the paretic limb is especially impaired where the paresis is due to cortical injury.

The Rolandic cortex is therefore spoken of as the motor cortex, and as the sensori-motor cortex indifferently; neither term is particularly helpful, inasmuch as all parts of the cortex that possess projecting fibres are both motor and sensory, whether they lie within the Rolandic region or not. In addition to the sensorial attributes of the Rolandic region, there seems to exist an area specially connected with cutaneous sensation in the gyrus fornicatus on the mesial aspect of the hemisphere. Regarding the relation between this area and the Rolandic area little definite is known, or on what principle they share, if they do, between them the sensations of the skin surface. It is noteworthy that, so far as known, cutaneous pain hardly if ever forms the aura in epilepsy.

The cortical field for vision is twofold—an occipital based on the retina, and a precentral based probably on afferent paths from the muscles moving the eyeballs and head. These two areas can be completely separated one from another by transections, without the reactions to stimulation of either being, so far as ascertainable, disturbed. Each has a projection system of fibres passing independently into the internal capsule, the one in front of the knee of the capsule, the other behind it. The dorsal portion of the occipital area seems connected with the upper part of the retina, the ventral portion with the lower part of retina, and almost the whole of each hemispheral field with the homonymous half of both retinæ, to judge by the movement which ensues, turning both eyeballs toward the crossed side. In accord with this result is the injury to vision caused by destruction of the occipital cortex, and especially of the cuneus and calcarine region. In the lower animals the cerebral hemispheres can be removed without abolishing all signs that the creature's reactions are influenced by difference between

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light and dark. In the case of man, destruction of the occipital region of vision causes hemianopsia to the degree of extinction in that part of the visual field of power to distinguish light from dark. The central region of vision is, in these cases, usually excepted from blindness, indicating that it has a bilateral cortical representation, the two retinal fields appearing, as regards this region, to be superposed in the visual cortex of each hemisphere. The dividing line is, with exception of the central vision region, a vertical one. The much heavier damage done to vision by cortical injury in man than in lower animals parallels the much heavier damage done to the movements of his limbs by similar injury in his case than in the case of lower animals. The double region, both occipital and precentral, playing by independent projection systems upon the same motor media, namely, third, fourth, and sixth cranials, in the case of the eyeballs serves to illustrate well the multiple control which must obtain also for the efferent cell groups in the spinal cord. Further, the perfect symmetry of the movements of the eyeballs evoked from the cortex illustrates well the extent to which, in many instances, each hemisphere controls mechanisms involving musculature in both halves of the body; other instances, besides the conjugate deviations of the eyes, are the movements of the neck, of the jaw muscles, of the larynx, of the tongue, and of the anal sphincter. In some of these movements the muscles thrown into contraction are the bilaterally symmetrical, *e.g.* in the larynx and sphincter; in others they are unsymmetrical, *e.g.* in the neck.

Damage to the first and second temporal gyri in man causes deafness in the opposite ear. Conditions of the ear which early in life produce deafness and deafmutism, result in consequent ill development of these gyri. The power of expression in spoken words is usually greatly disturbed by injury to the inferior frontal convolution of the left hemisphere. The muscles used in the act of speech are by no means paralysed; yet the existence of the corresponding area on the right hemisphere does not enable their control and use for speech. There is evidence which connects damage of the upper temporal convolutions with a condition in which, though general hearing is not lost, a spoken word fails to be intelligible. The patient cannot speak the word but understands it when it is shown to him in type or written. This is an *auditory aphasia.* A condition of alexia akin to this seems connected with lesion of the left angular gyrus—a kind of aphasia in which the patient cannot understand a written or printed word though he can hear it intelligently. Lesion interrupting the nexus between the visual and motor areas damages power to recall the name or use of an object when seen, but its name and use may return if it be handled—that is, when muscular and cutaneous impressions which it gives are added to the visual. The dog after cortical ablation loses fear of the whip; although he sees it he fails to recognise it.

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A cortical "centre" of smell is inferred chiefly from comparative physiology to lie at the tip of the temporal lobe, and to be closely connected with the hippocampal gyrus. Of the topography of gustatory mechanism in the cortex even less is known. Smell and taste are, however, senses which, in man, are probably relatively little developed in comparison with their potency in lower animals.

A great part of the cortex of the brain is called "inexcitable," that is, when examined experimentally no obvious reaction is provoked from it. This holds true of the great "association fields" (Flechsig) (Figs. 2 and 3). At birth few of the fibres of the cerebrum are myelinate. The sensory paths acquire myelin before the motor tracts. The paths from the roots of the spinal nerves are first to reach completion, the fibres for auditory impulses the latest. The Rolandic cortex or somæsthetic area, the occipital or visual, the superior temporal or auditory, and the uncino-hippocampal or gustato-olfactory, are all regions marked off from the rest of the cortex by early receiving myelinate paths from the sense organs. But around them, and forming in man the larger portion of the whole cortex, are regions less directly associated with the reception of sensory impressions. In these myelination of the nerve fibres does not for the most part occur until a distinctly later period, so that they are distinctly later in acquiring their structural perfection and functional activity. As myelination goes forward nerve fibres are traceable passing from the sense centres into these surrounding and intermediate fields; the intermediate fields interconnect the sense centres. Hence they are called associated centres. These form the great so-called inexcitable fields of the hemisphere, and they are, relatively to the rest of the cortex, far larger in the human brain than in the brain of the highest apes.

A portion of the "association" field of the cortex is the prefrontal area. Removal of one frontal lobe produces in the monkey and dog little obvious result—transient blunting of all sensations on the side opposite to the lesion, and some paresis of the muscles of the neck and trunk, which move these parts away from the side of lesion. These effects pass off, and if then the remaining frontal lobe be removed, not only do similar transient symptoms on the opposite side of the body, but a ceaseless wandering activity ensues, and the character of the animal appears to have suffered change. Fear and excessive irritability become predominant. Capacity to learn seems lost, and curiosity, affection, and pleasure, as far as these are signified by animals, disappear.

TROPHONEUROSIS

Intracranial section of the trigeminus is followed in many instances by keratitis, ulceration of lip, palate, etc. Broncho-pneumonia ensues

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and proves fatal after bilateral vagotomy. Severance of the nerves of a limb produces wasting of its muscles even of its bones and ligaments. Section of the spermatic nerve induces atrophy of the testis. Section of the nerves of the salivary glands results similarly in them. Most striking of all is the nutritive change ensuing in skeletal muscles after their separation from their nerve-centres. In two to three weeks' time their fibres begin to be measurably smaller than normal, and after some months they dwindle out of recognition, leaving a tissue in appearance like connective tissue. The rate of change differs with different muscles, and in different fibres of the same muscle. In the "red" muscles and in "red" fibres of muscles the atrophy is slower than in "pale" fibres. The muscle fibres inside the sensory organs of the muscle, the "muscle spindles," do not apparently atrophy at all. Accompanying this histological change are the well-known alterations in the electrical reactions of the muscle which constitute the "reaction of degeneration."

Injury to sensory as well as to motor nerves alters the nutrition of peripheral tissues. In the skin both atrophy and inflammation may occur secondarily to injury or disease of a nerve trunk. In *herpes zoster* the primary lesion seems an inflammatory condition of the spinal ganglion; on this ensues the eruption which occupies more or less of the field of skin for which the ganglion cells provide sensory channels. Recent research (Dr. Head) places beyond doubt the connection between this cutaneous eruption and focal inflammation of corresponding spinal ganglia. Again, when the sensory spinal nerve roots of a limb are cut proximal to the ganglia, there is distinct wasting in much of the musculature of the limb: ulceration is prone to occur over certain bony points of the limb. This ulceration tends to rapidly extend from the skin to the deep tissues (bed-sores).

Such instances as the above—and their number could be easily extended—yield evidence that every nerve and nerve centre possesses a so to say trophic influence on some tissue. Nor is this surprising. The sole function of a nerve is to influence the activity of the tissue which it innervates. If by "trophic nerves" be understood no more than that, all nerves are "trophic nerves." That, on the other hand, any nerve directly modifies the nutrition of a tissue independently of inducing vascular changes in it, or of influencing those particular metamorphoses of material for which the tissue is the machine specially adapted, there is no proof. The function and nutrition of these physiological machines form together an inseparable unity. This is a logical corollary to the "cell theory." Under the notion of normal nutrition is understood maintenance of a certain average form and certain average chemical composition. When the maintenance of the nutrition of one cell is greatly dependent on the maintenance of its connection with another cell, the inference amounts to certainty that the latter governs the activity of

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the function of the former. This consideration shows how greatly the skeletal muscles are governed by the spinal motor nerve cells. No other tissue except theirs practically dies down in consequence of destruction of the spinal cord.

The inflammation so prone to occur in exposed sensifacient surfaces when their nerve channels are broken stands in no such direct relation to the nerve injury. Anæsthesia, including analgesia, of the surface leads to unperceived accumulation of irritant effects. The most exposed parts ulcerate in consequence of long-continued and undue pressure. Trigeminal keratitis can be avoided by protection of the eye from dirt. The broncho-pneumonia after double vagotomy can be avoided by gastrostomy.

Not only are nerve cells trophic, in the above sense, for extrinsic tissues, but they are trophic for other nerve cells. The direction of the trophic influence is the same as that of normal nerve conduction. A series of many links in a nerve-cell system may be affected by destruction of the first member of the series, counting in the direction of normal conduction of nerve impulses—thus from afferent nerve cell along the cord to the brain and out by some efferent path. Thus destruction of the retina in early life induces atrophic changes in cells of the anterior corpus quadrigeminum, pulvinar, lateral geniculate body, and even cortex cerebri of the occipital visual region. Amputation of a limb leads to atrophic change in cells of the corresponding region of the spinal cord.

C. S. SHERRINGTON.

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DISEASES OF THE NERVOUS SYSTEM

The Neurone in its Relation to Disease of the Nervous System

In a modern text-book upon diseases of the central nervous system, the separation of those affections above the level of the foramen magnum into cerebral disorders from those below into spinal, no longer holds good. The present accepted conception of the constitution of the central nervous system is that of a series of neurones, some of which commence in the cerebrum and terminate in the spinal cord, while others, arising in the spinal cord and posterior spinal ganglia, terminate in the several portions of the brain. As many forms of organic nerve disease are at present construed as degenerations of one or other system of neurones, it is clear that no hard and fast line can be drawn between those disorders which are of cerebral as contrasted with those of spinal origin.

The anatomical structure and relations of the neurone or nervous unit have been described on a previous page, so that it will be only necessary in this introduction to refer to its bearing in connection with morbid processes and clinical types of disease. Speaking generally, the neurone consists of three parts: the nerve cell with its protoplasmic processes (dendrites), the axis-cylinder process (axone) and its end termination. The chief function of the cell is to maintain the nutrition of its processes, and to receive from and convey to its axis-cylinder process impressions from the terminal of an adjacent neurone. Although it was originally held that impressions were transmitted from one neurone to another by contact only, the most recent researches into the histology of nerve cells would appear to show that this is not universally applicable. Notwithstanding the probable existence of some continuity between the termination of one neurone and the cell of another so far as the conduction of nervous impulses is concerned, they appear to be in the main independent trophic units. In the study of cerebrospinal diseases and of the chronic degenerations which affect the nervous system, the trophic independence of the neurones may still be regarded as the most satisfactory theory which has yet been promulgated. Its appreciation makes intelligible certain facts which were previously difficult to comprehend. Such are the discrepancies which exist

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between the direction of conduction and of degeneration of sensory fibres, and the arrest of secondary degeneration in the anterior horns and other nuclei of gray matter, as well as the existence of certain paralytic affections without a discoverable lesion after death.

The neurones which form the central nervous apparatus conduct in two directions, towards and away from the cerebral cortex. Hence they may be primarily divided into afferent, or cerebri-petal, and efferent, or cerebro-fugal neuronic systems. These form the main channels for sensory and motor conduction. There are, however, numerous subsidiary neuronic systems having important functions; of these may be mentioned the cerebellar (spino-cerebellar and cerebello-medullary), the intra-cerebral, and those belonging to the sensory cranial nerves. It will be necessary to briefly refer to the chief neuronic systems of the cerebro-spinal axis and their peripheral extensions. They are—

(1) **The efferent neuronic systems.**—The efferent neurones are of two orders: the *cerebro-spinal* and the *spino-peripheral*. The former, having their cell bodies in the Rolandic area of the cerebral cortex, transmit their axis-cylinder processes through the centrum ovale, internal capsule, and pyramids to terminate in connection with the large ganglionic cells situated in the gray matter of the anterior horns of the spinal cord and its bulbar homologues, the nuclei of the motor cranial nerves. They form the well-known pyramidal system. The relations between the upper and lower efferent neurones in the spinal cord are not yet clearly defined. There is some evidence in favour of the interposition of "dendraxones" which transmit impressions from the pyramidal tracts to the anterior cornual cells (Von Monakow and Redlich). The latter, or spino-peripheral, have their cell bodies in the gray matter of the anterior horns and motor cranial nerve nuclei, and their axis cylinders form the motor fibres of the peripheral nerves—cranial and spinal. Their terminations are in the voluntary muscles (Pl. III. *a-b*, *c-d*).

(2) **Afferent neuronic systems.**—The first in order of these systems, commencing from the periphery, is that of the posterior root ganglia, the so-called posterior ganglionic system. Having its cell body in the posterior root ganglion, it forms the sensory portion of the peripheral mixed nerve, the posterior spinal root and the continuation of the root in the posterior columns of the spinal cord as far as the posterior columnar nuclei (nucleus gracilis and cuneatus). The second in order is the fillet system, which passes through the medulla and pons Varolii from the posterior columnar nuclei, where the cell body is located, to the opposite optic thalamus;

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while the third system extends from the optic thalamus, which contains the cell bodies, through both limbs of the internal capsule and the centrum ovale, to terminate over an extensive area of the cerebral cortex (Pl. III. *e-f*, *g-h*, *i-k*).

Subsidiary to these afferent systems are others, of which the chief are: (*a*) A columno-cerebellar, from the posterior columnar nuclei to the lateral lobe of the cerebellum, and (*b*) a cerebello-thalamic, from the cerebellum to the optic thalamus, the cerebral continuations of which latter are to be found amongst the thalamo-cortical fibres already mentioned.

(3) **Association neurones.**—Large numbers of short and long neuronic systems are to be found in all portions of the central nervous system. As these bring the several portions of, for example, the cerebral cortex into relation with each other and connect corresponding portions of the brain upon opposite sides, they may be described as association, internuncial, and commissural systems of neurones.

Degeneration of the Neurone

The neurone degenerates as a whole. The pathological changes affecting the cell and the axis-cylinder process are well marked, but the changes, if any, implicating the terminal have yet to be observed and studied. Long tracts of degeneration indicate implication of a neuronic system throughout its extent. By this means certain diseases have been found to select certain neuronic systems, so that a classification of many of the chronic degenerative affections may be made according as they are found to implicate the first, second, or other neurones of the afferent or efferent systems. These will be presently detailed. It is first necessary to briefly describe the pathological changes found in the neurone as a result of injury or disease.

(1) *The nerve cell.*—By aid of recent staining re-agents, more especially the Nissl method and its modifications, a ganglion cell is found to consist of a nucleus surrounded by a number of granules—the chromatic substance or Nissl bodies—which are continued into the protoplasmic processes, but not into the axis cylinder. Between the granules are clear spaces (Fig. 5, *A*). As the result of certain morbid states, whether of toxæmic nature or as a result of impaired blood supply, the ganglion cell undergoes several characteristic changes. There is first a swelling and excentration of the nucleus, then swelling and disappearance of the chromatic bodies (Fig. 5, *B*),

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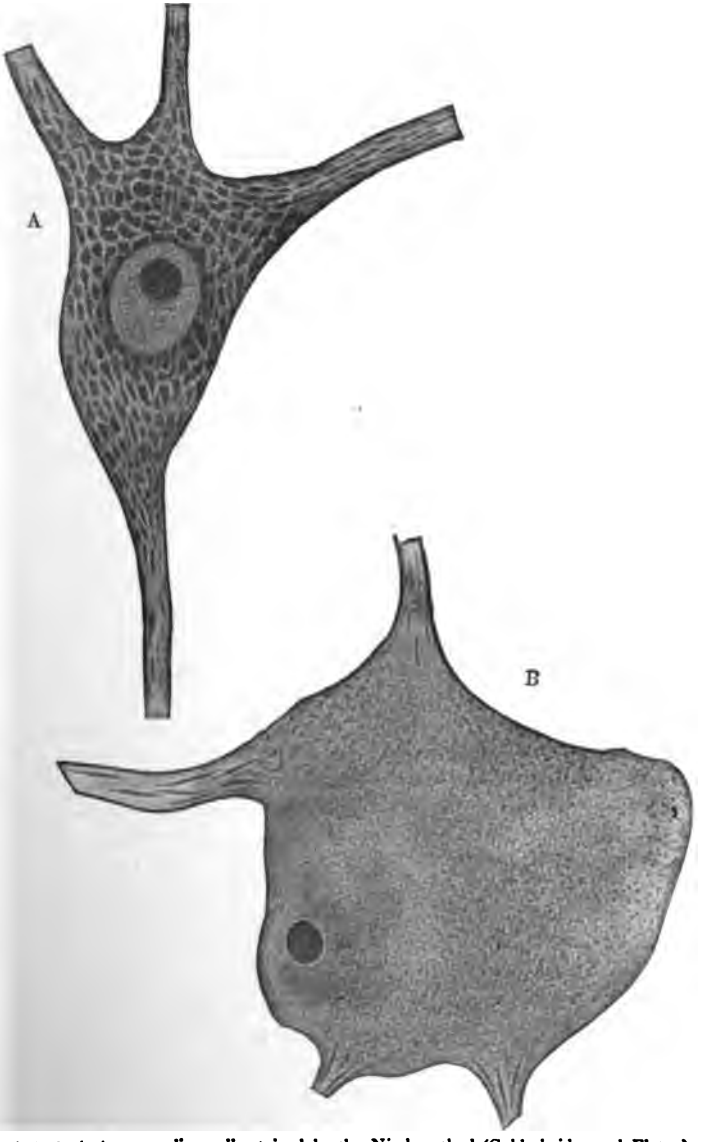

FIG. 5 represents two ganglion cells stained by the Nissl method (Goldscheider and Flatau). *A* depicts the appearances consistent with health: the nucleus well defined and clear, the "Nissl bodies" or chromatic substance of normal arrangement. *B* is a ganglion cell undergoing degeneration after amputation of a limb; the nucleus is excentric and ill defined, the cell is swollen, the "Nissl bodies" undergoing fatty degeneration, while the cell processes are broken off.

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their replacement by fatty debris, and breaking off of the cell processes, a shrunken and sclerotic cell eventually taking its place. Should this occur, death of the cell has ensued, but it would appear as if recovery were possible should disintegration be arrested before the onset of fatty degeneration. The nerve fibre undergoes simultaneous alteration, breaking up of the myeline sheath, with swelling and atrophy of the axis cylinder.

Other but less well understood changes are seen in vacuolation, pigmentation, and calcification of ganglion cells.

(2) *The axis-cylinder process.*—Secondary (Wallerian) degeneration. When the neurone is broken by severance of the axis-cylinder process, changes at once begin to take place on both sides of the lesion. In the distal part these consist of swelling and breaking up of the myeline sheath and fatty degeneration, and a varicose appearance of the axis cylinder. In course of time the debris becomes absorbed, and a sclerotic condition from neuroglial overgrowth is produced. Associated with this change is a disintegration and disappearance of the terminal, so that in the gray matter there is an absence of the fibrillary network, with clear spaces surrounding the ganglion cells.

But the portion of the fibre attached to the cell also undergoes change. This is of the nature of a degeneration, and implicates the cell from which the fibre takes origin. It is a gradual atrophic process, reaching its highest development some time after the infliction of the lesion.

A form of simple atrophy of nerve cells and fibres without any structural alteration has been described. It is seen in the neuronic systems next in order to those which have been primarily destroyed. Such, for example, are the atrophic changes found in the spinal cord after amputation; or in the mesial fillet after lesions of the cerebral cortex. This atrophy establishes the possibility of alteration in one series of neurones, when deprived of the normal impulses from adjoining systems, even though the intervening nerve cell is unaffected.

THE IMMEDIATE CAUSES OF DEGENERATION OF THE NEURONE.—The changes above described may result from many and various causes, the chief of which is no doubt toxic material circulating in the blood. This implicates the neurone either directly, or indirectly by the production of degenerative changes in the walls of the arterioles. The deleterious influence of certain chemical poisons upon nervous tissue has long been known; and important facts are being gathered bearing upon the baneful influence which other toxins of known or merely surmised bacterial origin exert; but

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we have yet to learn what is the materies morbi influencing the neurone in many cases of so-called functional disease; and how far the neurone may undergo disintegration consistent with a return to health.

Of chief importance is (*a*) the syphilitic poison. This produces its effects either indirectly, through characteristic vascular changes, elsewhere described, or directly, by selecting certain neuronic systems, as is seen in tabes dorsalis (locomotor ataxy) and general paralysis of the insane. (*b*) The poisons of the acute specific fevers appear to implicate the neurone indirectly through their influence upon the blood-vessels. Amongst these may be included influenza, which appears to have a peculiarly deleterious effect upon nervous tissue, and malaria, both acting chiefly through the production of vascular thrombosis. (*c*) Anæmic states of the blood exert a malign influence, which is especially well seen in the degenerations associated with pernicious anæmia. Under this head may also be included altered blood states, associated with prolonged pyrexia and hyperpyrexia. (*d*) Toxic blood states, as they are exemplified by diabetes, leukæmia, pellagra, and beri-beri. (*e*) Chemical poisons, such as alcohol, lead, silver, mercury, etc. (*f*) There is reason to suppose that poisons produced within the body (auto-intoxication) may lead to degeneration of the neurone. This would appear to be especially a cause of some acute mental disorders. (*g*) Trauma. The influence of trauma, although not clearly defined, may be productive of degeneration of the neurone. Under this term may also be included severe forms of sunstroke.

Degenerations classified according to the Neuronic System primarily involved

I. *The cerebro-spinal or upper efferent neuronic system, commonly called the pyramidal system* (Pl. III. *a-b*).

The degenerative affections of this system are—

(1) The spastic variety of general paralysis of the insane.
(2) Primary and secondary lateral sclerosis.
(3) Old-standing hemiplegia, whatever its causation.
(4) Spastic paraplegia and diplegia of children.
(5) The paraplegic type of disseminated sclerosis.

II. *The spino-peripheral or lower efferent neuronic system* (Pl. III. *c-d*).

The diseases included under this heading are—

(1) All forms of peripheral neuritis—local or multiple.

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(2) The effects of acute anterior poliomyelitis and its analogues, acute bulbar paralysis and acute ophthalmoplegia.
(3) Acute ascending or Landry's paralysis.
(4) Chronic anterior poliomyelitis, *i.e.* progessive muscular atrophy, progressive bulbar paralysis and ophthalmoplegia externa.

I. and II. *Simultaneous affections of both the efferent neuronic systems*:—

(1) Amyotrophic lateral sclerosis, a disease in which there is degeneration of the whole motor mechanism, from the cerebral cortex to the peripheral end organs in the muscles. Fig. 20, p. 163.
(2) The advanced stage of general paralysis of the insane.

III. *The lowest afferent or posterior ganglion neuronic system* (Pl. III. *e-f*).

This system is affected by two characteristic disorders—

(1) Tabes dorsalis (locomotor ataxy). Fig. 17, p. 162.
(2) The ataxic form of general paralysis of the insane.

There is no degenerative disease limited to the fillet and thalamo-cortical neuronic systems, although any lesion, whether vascular or other, interfering with the continuity of these systems, leads to their secondary degeneration.

A few diseases would appear to simultaneously implicate the lowest afferent and the upper efferent neuronic systems. Of these the most important are—

(1) Friedreich's ataxia. Fig. 19, p. 163.
(2) The combined postero-lateral sclerosis, which is seen in some forms of pernicious anæmia and also as a sub-acute primary disease. Fig. 18, p. 162.
(3) The advanced stage of disseminated sclerosis. Fig. 21, p. 165.

The General Symptomatology of Disease of the Chief Neuronic Systems above described

Without entering into any detailed description of the diseases just mentioned, the statement may be made that certain general clinical phenomena are characteristic of the affections of individual neuronic systems, by which their several diseases may be recognised and referred to their proper system; and these are given in the following tabular form:—

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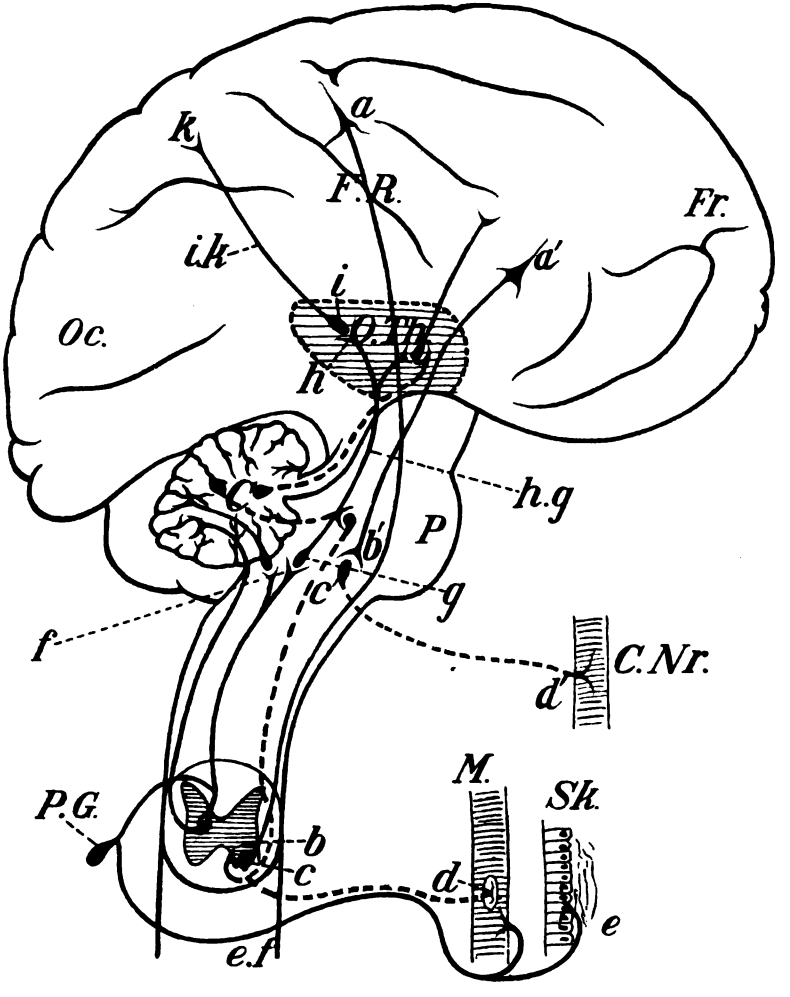

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PLATE III

This is a schematic representation of the chief afferent and efferent neuronic systems affected by disease; and has to be studied in connection with the descriptions on pp. 39 to 41 (W. A. Turner).

Red continuous lines = first efferent or cortico-spinal neurones: *a-b*, cortico-spinal, or pyramidal neuronic system; *a'-b'*, cortico-bulbar neuronic system. Red interrupted lines = second efferent or spino-peripheral neuronic systems: *c-d*, spino-peripheral system; *c'-d'*, bulbo-peripheral system.

Blue lines = spino-cerebral afferent neuronic systems: *e-f*, first or lowest afferent system or system of the posterior spinal ganglion; *h-g*, middle afferent or fillet system; *i-k*, highest afferent or thalamo-cortical neuronic system.

Green lines indicate the cerebellar (direct and indirect) neuronic systems. Green continuous line = spino-cerebellar systems; green interrupted line = cerebello-spinal systems.

FR, fissure of Rolando; *C. Nr.*, motor cranial nerves; *M*, body musculature; *Sk*, skin; *P*, pons Varolii; *Pg*, posterior root ganglion; *C*, cerebellum; *O. Th.*, optic thalamus.

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PLATE IV

FIG. 1.—A diagrammatic representation of the tracts of the spinal cord. (Modified from Lenhossek by W. A. Turner.)

The blue coloration indicates the portion of the posterior spinal ganglion system, occupying the posterior columns, No. 7.

The red, the pyramidal system: 1, direct pyramidal, and 5, crossed pyramidal tracts.

The green indicates cerebellar afferent systems: 3, ascending antero-lateral tract system of Gowers; 4, direct cerebellar system of Flechsig. The stippled green indicates the descending medullary and indirect cerebellar connections: 2, descending antero-lateral system; 2′, encephalo-spinal fibres (other than cortical).

No. 6 is the "lateral limiting layer"; No. 8, Lissauer's tract; No. 9, septo-marginal tract; No. 10 is the comma tract of the posterior column.

FIG. 2 shows the termination and commencement of the chief neuronic systems at a given level of the spinal cord. (Modified from Lenhossek by W. A. Turner.) The colouring is in harmony with Plates III. and IV. fig. 1. The figuring is the same as in Plate IV. fig. 1.

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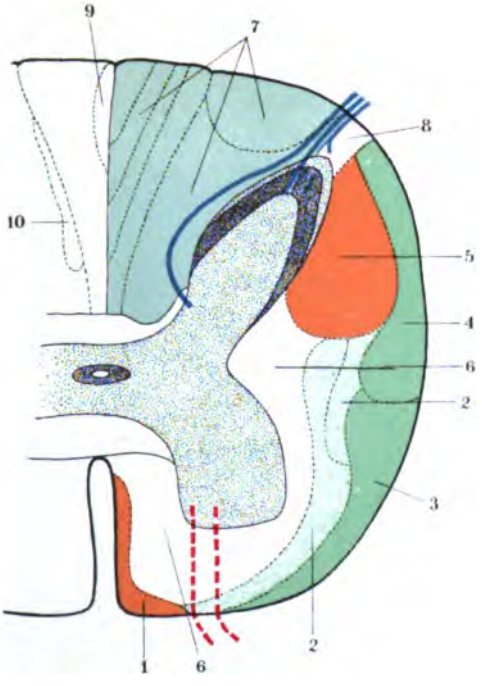

FIG. 1.

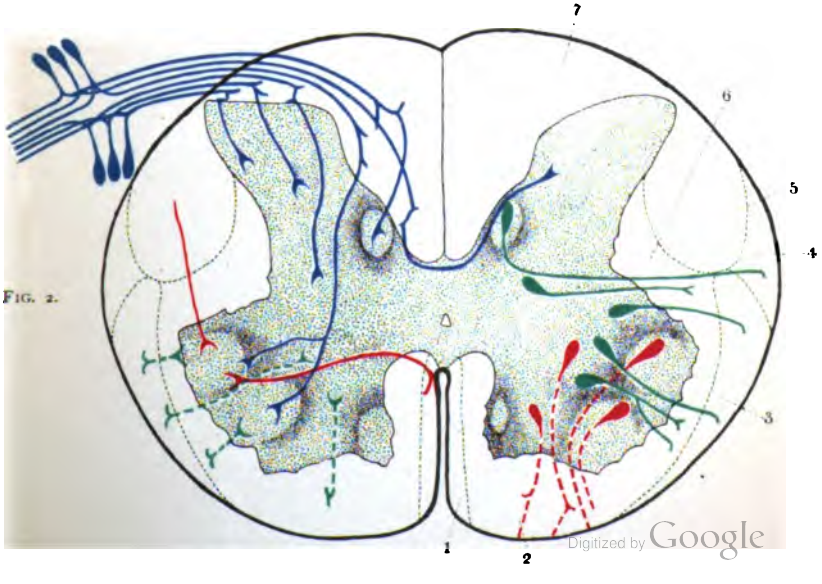

FIG. 2.

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I. *Symptoms of disease of the upper efferent or cerebro-spinal system* (Pl. III. *a-b*).

(1) Paralysis or defect of motor power.
(2) Muscular rigidity and a spastic gait.
(3) Increase of the deep reflexes: exaggerated knee-jerks, the presence of an ankle-clonus, and the extensor type of plantar reflex.
(4) Absence of muscular wasting other than can be accounted for by disuse of the limbs.
(5) No impairment of sensation.
(6) No alteration in the electrical excitability of the muscles, except perhaps a slight quantitative increase or decrease.

II. *Symptoms characteristic of affections of the lower efferent or spino-peripheral system* (Pl. III. *c-d*).

(1) Motor paralysis or paresis according to the severity or extent of the lesion.
(2) In old-standing cases, contractures dependent upon greater implication of certain muscular groups.
(3) Atrophy of the muscles.
(4) Abolition of the deep reflexes.
(5) Loss or defect of faradic excitability, and a qualitative alteration in the galvanic excitability of the muscles; the reaction of degeneration.
(6) Slight sensory impairment from simultaneous affection of sensory nerve fibres.
(7) A tendency to "trophic" phenomena, such as glossiness of the skin and local increase of perspiration.

There exists a slight difference in the symptomatology, according as the anterior horns (anterior poliomyelitis) or the peripheral nerves (neuritis) are originally affected. Nos. 6 and 7 are not seen in poliomyelitis.

III. *Symptoms of degeneration in the lowest afferent or posterior ganglion system* (Pl. III. *e-f*).

(1) Absence of motor paralysis.
(2) The presence of various degrees of ataxia.
(3) Anæsthesia, paræsthesia, pains and delayed conduction of sensation.
(4) Abolition of the knee-jerks.
(5) Various associated phenomena, such as the loss of the pupillary light reflex, gastric and other crises, retention of urine, perforating ulcer and osteo-arthritic affections.

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(6) Absence of impairment in the electrical irritability of the muscles.

General Etiology of Diseases of the Nervous System

The results of investigation into the pathology of the diseases of the nervous system show that in their general causation, their structural modifications, and their possible prevention, organic nervous disorders do not materially differ from those of the other organs of the body. It may be accepted as a general principle that the same factors are at work in the production of disease, whether the tissues affected be those of the nervous, or any other system, and of these the most important are: an inherited disposition, whereby the nervous tissue is rendered intrinsically less antagonistic to the ordinary external causes of disease; and a certain accessibility to the effects of infective or toxic agents carried thereto by the blood and lymph streams.

Heredity.—The influence of an inherited disposition to nervous disease is manifested in the production of functional disorders and the neuroses, such as insanity, idiocy, epilepsy, alcoholism, and migraine, rather than by gross organic conditions, such as tumour, encephalitis, and sclerosis. But in some forms of spinal disease, such as hereditary ataxia, hereditary spastic paraplegia, and some forms of muscular dystrophy, the evidence of heredity is obvious, a well-defined form of the disease reproducing itself from generation to generation; while a further evidence of an inherited neuropathic disposition is illustrated by the occurrence of insanity and epilepsy in the family or its collaterals. Evidence of heredity is also to be found in the transmission of the neuropathic constitution, where, in the family history, functional nervous disease of various kinds is known. Of what the neuropathic constitution consists, there is no clear evidence, but it probably indicates a nervous system more than ordinarily accessible to the external causes of disease in general. This want of resisting power on the part of the nerve elements may be the expression of an inherent nervous instability, or the effect of such causes as alcohol or syphilis in one or both parents. The tendency for death to occur in certain families from cerebral hæmorrhage is due rather to a predisposition to vascular or renal degeneration than to any transmitted instability of the nervous system. The influence of inherited syphilis in the causation of nervous disease is not fully known. Apart from the vascular lesions in infancy and childhood, of which

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it is the direct causation, syphilis is largely instrumental in the production of some forms of backwardness and defective development, imbecility, epilepsy, and juvenile general paralysis.

SEX AND AGE.—The influence of sex and age as factors in the causation of organic nervous disorders is chiefly determined by the nature of the infective diseases to which the sexes or different ages are more prone. Thus males are more liable to those conditions brought about by traumatism, by syphilis, and by chemical poisons; children to the sequelæ of the acute exanthemata, diphtheria, and allied affections; adults to the influence of syphilitic infection, influenza, and alcoholism; while the old are specially liable to the disturbances which follow in the wake of vascular degenerations.

TRAUMATISM.—Injury may be productive of nervous disease in two ways, either directly, as an excitant of local lesions, such as fracture of the skull, giving rise to contusion of the brain, hæmorrhage, or localised inflammation, and fracture-dislocation of the spinal vertebræ, producing myelitis or injury to the nerve roots; and, indirectly, as a cause of general nervous disorders unassociated with obvious organic alterations. Whether injury may act as the direct cause of degeneration of the neurone, leading to various forms of sclerosis, is not at present clear, nor is the experimental evidence on this point sufficiently proven. The influence of traumatism, however, upon already existing nervous disease is important. If there be already an organic nervous malady, injury invariably intensifies it, while its influence upon pre-existing vascular disease in the production of apoplexy is great. Some of the cases of so-called spinal concussion or "railway spine," commonly seen after railway accidents, are now regarded as indicating a cerebral condition allied to neurasthenia.

POISONS—INFECTIVE.—*Syphilis* is an important cause of organic nervous disorders. It produces its effects in one of two ways. First, by the formation of obliterative changes in the lumen of the cerebral arterioles, leading to thrombosis, or by small-celled infiltration of the adventitia of the blood-vessels, leading to gummatous tumour formations. Secondly, through the influence which it exerts in the production of chronic progressive degenerations. The first series of lesions may appear at any time, but are usually early manifestations of the disorder, the greater number of cases, according to one authority, happening during the first three years after infection. These conditions are relatively frequent, and are illustrated by gummata, thrombosis, both spinal and cerebral, meningo-encephalitis and meningo-myelitis, and by local meningeal inflammation, causing paralysis of the cranial nerves.

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To the degenerative disorders the term "para-syphilitic" has been applied. These make their appearance at a relatively late period after infection, usually from the fifth to the twentieth year, the best known examples being tabes dorsalis or locomotor ataxia, and general paralysis of the insane. Although acquired syphilis is a common cause of these changes, the inherited disease is not without its malign influence upon the central nervous apparatus, for from this cause there may result both the vascular and the degenerative varieties.

Infection other than syphilis. — Here are included all those pathogenetic conditions, whether of recognised, or merely surmised, bacterial causation, which lead to nervous disorders, or which produce nervous symptoms as sequelæ: such are influenza, typhoid fever, the exanthemata, diphtheria, etc. Such forms of infection produce their results either through the influence of micro-organisms, or more commonly through the agency of their products carried by the blood and lymph streams to the nervous tissues; and their effects are either directly upon these or indirectly through vascular lesions, as are seen in acute inflammatory, thrombotic, and hæmorrhagic conditions.

Infection does not necessarily occasion only acute disease of the nervous system; subacute and even chronic affections may be so produced, such as disseminated encephalitis and multiple sclerosis. At the risk of forcing the infective theory of the causation of nervous disorders to an unnatural extreme, one is nevertheless inclined to refer the chronic degenerative diseases—disseminated sclerosis, bulbar palsy, amyotrophic lateral sclerosis, and ophthalmoplegia—to a possibly toxic origin, inasmuch as it is not uncommon to find arterial dilatation, degeneration, and other evidence of chronic inflammation on microscopic examination. There is certainly good reason to suppose that an acute infection may aggravate a pre-existing nervous disorder.

Tuberculous affections of the brain and its membranes are usually the result of secondary infection, the primary source of the disease being found in other parts of the body.

It seems scarcely necessary to do more than mention direct bacterial infection as the cause of acute suppurative conditions of the brain and its membranes and of sinus thrombosis. Apart from the bacillus of tubercle, the micro-organisms most frequently found are the staphylococcus and streptococcus pyogenes, and the diplococcus pneumoniæ.

A source of infection not commonly considered, but long known,

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which may explain numerous cases of so-called idiopathic disease, is probably to be found in the absorption of toxic substances from the alimentary canal. The influence of this in the production of acute mental phenomena is recognised, but it is not yet clear how far organic cerebral disease may be caused in this way.

TOXÆMIC.—Another toxic cause of morbid nervous phenomena is to be found in modifications of the internal secretions. To this are to be attributed the nervous symptoms met with in such diseases as myxœdema, cretinism, and exophthalmic goitre. In this group are also to be placed the nervous manifestations associated with diabetes.

Very definite degenerative changes are found in the spinal cord as the result of severe anæmias, and also of certain food poisons—ergotism, pellagra, and lathyrism (see Vol. II. p. 45).

CHEMICAL POISONS.—A considerable number of nervous affections are due to the influence of chemical poisons. It is well known that the direct influence of these substances is chiefly upon the peripheral nervous apparatus, as may be seen in the paralyses following prolonged use of alcohol, lead, arsenic, mercury, and others. The cerebral affections caused by the prolonged use of chemical poisons are more commonly of a mental character, rather than of an organic nature, such as are described in this chapter. In the same category may be placed the cerebral phenomena associated with beri-beri.

VASCULAR SUPPLY.—Although the quality of the blood supplied to the brain and cord is a more frequent cause of disease than are alterations in quantity, yet the latter is frequently responsible for symptoms even of a serious character. Prof. Hill has shown that the total quantity of blood within the brain is liable to but very slight variation, although considerable difference may exist in the proportion in which this quantity is distributed between the arterial and venous sides of the capillaries. Whether an excess of arterial supply (hyperæmia) ever exceeds normal limits seems to be doubtful, since among other reasons there appears to be no effective vaso-motor control of these vessels, and for the same reason there is probably no spasm of the cerebral arteries leading to anæmia. A venous engorgement (passive congestion) may, however, occur either suddenly and be of transient duration, as in straining, prolonged holding of the breath, severe coughing, epileptic convulsions, etc., or more slowly, as by compression of the jugular veins, or extreme obstruction in the right heart. The increased venous pressure thereby induced will lead to cerebral anæmia—so far as the due

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supply of arterial blood is concerned—a condition that is also induced by a fall in arterial pressure, as from an embolus, from cardiac failure, from cerebral hæmorrhage, from extreme determination of blood to the splanchnic area, or from compression of the carotid arteries. A diminution of the blood supply would be slowly established by changes in the vessel walls tending to narrow their calibre, such as atheroma, or endarteritis obliterans. These changes will of themselves favour the occurrence of thrombosis or of hæmorrhage.

In conclusion, there are certain conditions which clinical experience indicates as causes, or at least determinants, of nervous disease, but of which the pathogenetic significance is not clear. Such are, exposure to cold and damp, as illustrated by myelitis and peripheral facial paralysis; the influence of overwork and mental and moral stress, worry and anxiety; while sexual excess or abuse is often accredited with an altogether undue significance.

As our knowledge of the pathology of nervous disorders becomes more complete, the list of so-called idiopathic diseases progressively diminishes; in fact, it is doubtful whether any organic nervous disorder may be rightly termed idiopathic. The methods at our disposal for investigating the finer changes occurring in the neurone point to a toxic influence as the commonest cause in the production of nervous disease. Until our knowledge, however, of the morbid changes underlying the insanities, epilepsy, convulsions, and coma is further matured, it would be premature to generalise on this subject.

W. ALDREN TURNER.

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ORGANIC DISEASES OF THE BRAIN AND ITS MEMBRANES

General Symptomatology

Although the morbid conditions affecting the nervous system are varied and numerous, the symptoms by which they manifest themselves are relatively few. In the broadest sense they may be divided into two series: the *subjective*, or those recognisable only by the patient; and the *objective*, or those obvious to, or detectable by the physician. As a general rule, in the nervous system symptoms depend more upon the locality of the disease than upon the morbid process. Thus, paralysis in its most general sense may arise from lesion of the motor mechanism anywhere between the cerebral cortex and the muscles at the periphery; but the result is paralysis, whether the cause be hæmorrhage, thrombosis, tumour formation, inflammation, or degeneration.

Paralysis.—By this term is usually meant loss or defect of voluntary motor power. Defect of motor power may be produced by arthritic adhesions, pain on attempts at movement, or by muscular spasm. True paralysis may be of several types: the hemiplegic, in which one side of the body is paralysed; the paraplegic, in which the legs are mainly affected; the diplegic, in which the movements of the arms as well as the legs are impaired; and the monoplegic, in which only one limb is materially involved. Various forms of multiple paralysis may also be met with; while single or local palsy is seen in affections of individual muscles, such as the external rectus, or of a group of muscles supplied by a single nerve, such as the facial.

Paralysis from organic cerebral causes, however, may be simulated by functional paralysis, the chief of which is the so-called "hysterical hemiplegia." In this the face is rarely implicated; the leg is often more paralysed than the arm, and there are usually the characteristic sensory disturbances elsewhere described.

Associated with the paralytic condition are various characteristic secondary phenomena. Of these the chief are — (*a*) *Rigidity of the muscles*, whereby the limbs assume awkward positions. Of the two varieties of rigidity, as seen, for example, in hemiplegia of cerebral origin, the late, or secondary form, is the more obtrusive. It

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is due to the presence of degeneration in the crossed pyramidal tract, the result of the primary lesion. It is a marked feature of old-standing cases of hemiplegia.

(*b*) *Post-hemiplegic disorders of movement.*—These are of various forms, but one of the most frequent is athetosis, which occurs most commonly in infantile hemiplegia. A variety of irregular movement on volitional effort has received the name of post-hemiplegic chorea, from the similarity of the jerking to that seen in chorea.

Crossed paralyses, by which is meant paralysis of a cranial nerve on one side and of the limbs upon the opposite side, are characteristic of lesions of the hind brain. They are of various kinds, and are described under lesions of the pons Varolii (p. 134).

CONVULSIONS AND SPASM.—A convulsion is an excessive discharge of nerve energy, and is commonly of two kinds: the *tonic*, in which the muscular contraction is maintained and tetanoid in character; and *clonic*, in which there is an alternate contraction and relaxation of the muscular fibres. Convulsions are usually general, involving the whole of the musculature; or they may be only unilateral or even implicate merely one limb or a part of a limb. To the latter the term spasm or mono-spasm is more usually applied. Convulsions are of cerebral origin, and arise either from irritative organic cortical disease, or from toxic causes. They form, in their most typical characteristics, the main features of epilepsy. Local spasm is not necessarily associated with any defect or loss of consciousness; it may, on the other hand, form the aura or commencement of a general convulsion, in which consciousness is entirely obliterated. A localised or unilateral convulsion is usually followed by temporary paralysis of the limb or limbs convulsed; while a general convulsion, as in severe epilepsy, may result in temporary general exhaustion and prostration. Convulsions arising from organic cerebral disease are characterised by a local commencement, such as the hand, thumb, or fingers, or the foot: a fact which emphasises the importance of ascertaining the exact mode of onset of all convulsive seizures, whether local or general.

LOSS OF CONSCIOUSNESS. — This is a common symptom of cerebral disease, and presents various grades of intensity; such are profound *sleep*, from which a person may be awakened; *lethargy*, from which he may be temporarily aroused; *stupor*, a state in which, with apparent obliviousness of surroundings, the patient may be aware of passing events; and *coma*, in which there is profound abolition of consciousness, with stertorous breathing and a tendency to death by asphyxia. The natural termination of many acute

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cerebral disorders is in coma. Thus an epileptic convulsive seizure invariably ends in this way, thereby denoting temporary, complete, and universal paralysis of the higher centres; in cerebral hæmorrhage, all the stages of interference with consciousness, from slight blunting of intelligence to complete coma, may be observed. In many forms of insanity, and more especially in general paralysis of the insane, the picture of a gradually invading coma is striking. In alcoholic intoxication also it is not difficult to study a similar sequence of events. The differential diagnosis of the several causes of coma met with in clinical medicine is important, and this is discussed later, on p. 71.

In severe cases there is complete muscular flaccidity; the pupils are usually dilated, and inactive to light, and the knee-jerks may be abolished; the breathing is stertorous and the patient dies from failure of respiration.

HEADACHE. — Headache is more commonly associated with general and local extra-cranial conditions than with serious organic cerebral disease. The common forms of headache are seen more especially in neurasthenia, in neuralgia, in migraine, in association with errors of refraction, in mastoid and aural disease, in anæmia, and renal disorders, and as a reflex symptom of gastric and uterine disease.

The principal cause of headache in organic cerebral disease is intra-cranial tumour. Its position is not necessarily dictated by the locality of the new growth; it may or may not be associated with general scalp tenderness, but sometimes tenderness on deep pressure may be elicited over the site of a tumour. The chief significance of headache in organic cerebral disease lies in its association with optic neuritis and vomiting; with hemiplegia and paralysis of one or more cranial nerves; or with evidence of the presence of meningitis.

VERTIGO, or giddiness, is a symptom commonly seen in cerebral disease. It is, however, so frequent in other conditions that its clinical significance, unassociated with other obvious symptoms of organic disease, is of little value. It is the most prominent feature of auditory and labyrinthine disease; it may be due entirely to ocular deficiencies; it is a common feature of epilepsy, while as a symptom of organic cerebral disorder it is most pronounced in disease of the posterior cranial fossa.

DELIRIUM is a frequent symptom in the later stages of organic cerebral disease. It is an acute mental condition, characterised by delusions and hallucinations, with confused recognition of objects

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and persons as such, and it is often accompanied by active efforts, chiefly the result of the disordered mental impressions. Apart from inflammatory conditions, such as meningitis, delirium is frequently due to pyrexia at the onset of the acute specific fevers, while it is the leading symptom in the acute alcoholic condition of "delirium tremens." In the diagnosis of cerebral disorders, its value is estimated chiefly by the association of the other and characteristic symptoms already detailed.

HOMONYMOUS HEMIANOPSIA is the term applied to blindness of

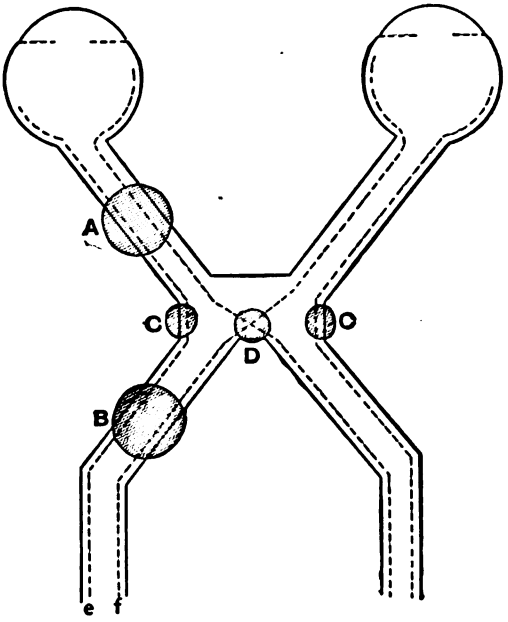

FIG. 6 is a schematic representation of the retinæ, optic nerves and tracts (after Hamilton), showing the arrangement of the crossed (*f*) and direct (*e*) visual fibres. *A* is a lesion of the optic nerve, which would cause blindness in the corresponding eye; *B* is a lesion of the optic tract, which would cause homonymous hemianopsia, or blindness of the half-fields to the opposite side; *C* is a lesion which would cause, if on one side, nasal hemianopsia in the eye on the same side; if bilateral, binasal hemianopsia; *D* is a lesion interfering with the decussating visual fibres and causing bitemporal hemianopsia.

the corresponding halves of both retinæ, and is due to interruption of the visual fibres in the optic tract, basal ganglia and optic radiations, and destruction of the cortical visual centres in the occipital lobe (Fig. 6).

The line of demarcation between the seeing and the blind

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halves of the retina may or may not pass through the fixation point, which corresponds to the macular region of the retina. If the fixation point is not divided, it is always included in the seeing, not in the blind half. There may exist various individual differences in the position and course of the dividing line. In some cases it is vertical, in others oblique; or it may be straight or irregular. Although complete homonymous hemianopsia is the rule, several irregular and incomplete forms have been observed; thus a quadrant hemianopsia has been described in which correlated retinal quadrants have been rendered blind. There are also various forms of irregular hemianopsia, giving peculiar perimetric tracings, which cannot be put down to any definite lesion, but are probably due to local inflammatory or other destructive change in the retina, optic nerve or tract.

In the majority of cases hemianopsia is absolute, *i.e.* there is loss of colour, form and light senses; there are, however, cases in which the sense of colour is chiefly impaired; this receives the name of *hemichromatopsia*.

Homonymous hemianopsia, occurring without any other associated localising symptom, is due to destructive lesion of the half-vision centre in the occipital lobe, or of the subjacent optic radiations. Right-sided hemianopsia may or may not be associated with word blindness or word deafness (*see* Aphasia). If such symptoms co-exist, the combination shows a concurrent affection of either the angular gyrus or the first temporal gyrus. When hemianopsia is associated with hemiplegia and hemianæsthesia, or with the latter alone, the lesion may with confidence be placed in the posterior limb of the internal capsule (p. 127, Fig. 12). Lesions of the basal ganglia—external geniculate body and optic thalamus—are also followed by hemianopsia, as is also destruction of the optic tract. The *hemiopic pupillary reaction* is supposed to be characteristic of a lesion of the optic tract. This consists in contraction of the pupil, when a ray of light is thrown upon the seeing halves of the retina, while no such reaction occurs when the light is turned upon the blind halves.

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DISEASES OF THE CEREBRAL MEMBRANES

Both the cerebral membranes are liable to diseased processes, but those of the inner or pia-arachnoid are the more frequent, as well as the more important. A few words only need be said upon the affections of the dura mater.

Pachymeningitis Externa

An inflammation of the external surface of the dura mater may be found resulting from injury to the skull, and under septic conditions may proceed to suppuration, the symptoms of which are those of cerebral compression. The outer surface may also be involved by an extension of erysipelatous or other inflammatory conditions of the scalp.

Chronic external meningitis, evidenced by adhesion between the dura mater and the inner table of the skull, is not uncommon on post-mortem examinations; it is found chiefly in the old in cases of chronic insanity, in epilepsy, and in the alcoholic. A localised adhesion is also found as the result of injury.

Pachymeningitis Hæmorrhagica—Dural Hæmatoma

This condition has received two names, as it was for long a matter of dispute whether the pathological appearances were the result of inflammation or hæmorrhage. All recent investigation supports the latter view. It occurs as a thickening upon the cerebral surface of the dura mater, due to the superimposition upon it of one or more layers of a fibrinous material, the number of layers depending upon the number of previous hæmorrhagic effusions which have undergone organisation. The false membrane may be stripped off in recent cases, leaving the shining dural surface underneath, or, in other cases, owing to a want of organisation of the clot, a cystic condition is seen at the autopsy. It is met with in the subjects of chronic insanity and in general paralysis of the insane. It is doubtful whether it occurs as an independent affection in the adult.

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The **symptoms** by which its existence may be distinguished from those of the insanity with which it is associated are the presence of convulsions at recurring intervals. These are either of a general or local epileptiform character, ending in coma, from which recovery may or may not be complete. Should recovery occur some amount of paralysis usually results. On the other hand, if the coma be slight, rigidity of the limbs and clonic muscular twitchings may be observed. As the disease progresses, the patient between the seizures shows increasing mental failure, partial paralysis, disturbances of speech, and eventually paralytic dementia. Death, as a rule, occurs in a convulsion or in the subsequent coma. The symptoms may be unilateral or bilateral; if the latter, there is a preponderance of symptoms on one side.

Diagnosis.—During the existence of the coma there are no means of distinguishing this condition from those other cerebral disorders characterised in their acute stage by loss of consciousness. It is only by a careful study of the whole course of the disease that dural hæmatoma may be differentiated. The disease is usually chronic, lasting sometimes several years, and presenting at intervals the convulsions of an epileptiform character which have been described.

The **prognosis** is essentially unfavourable. Each succeeding apoplectiform attack usually renders the patient mentally feebler, until a condition of profound dementia supervenes. Death is apt to occur during a seizure, being preceded, as is usual in such conditions, by considerable elevation of temperature.

As regards **treatment**, little of value can be done. During the apoplectiform seizure the procedures and remedies described later under cerebral hæmorrhage should be applied and administered. Trephining after the attack, with a view to removal of the clot, is not likely to be of much service, as the effusion is usually extensive, and in many instances bilateral. During the intervals large doses of the iodides may be given with a view to hasten the absorption of the clot.

LEPTOMENINGITIS

Varieties.—The several forms of meningitis may be classified in a variety of ways. First, there is the old method based upon the rapidity of the disease, and termed *acute* or *chronic*, according as it runs a swift or a slow course. Secondly, there is a classification founded upon the region of the brain chiefly affected, on which are based such terms as "convexity meningitis," "basal meningitis,"

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"ventricular meningitis." These terms are of little practical value, as meningitis starting in any locality tends to become general.

The modern and scientific method is that framed upon the view that the disease depends upon bacterial infection. In this way there are differentiated: (1) *Pneumococcus meningitis*, characterised by the presence of the diplococcus pneumoniæ. This is met with either as a primary infection, or as a complication of lobar pneumonia and endocarditis. It may also be secondary to a suppurative otitis media. (2) *Streptococcus meningitis*. Although the streptococcus has been found alone in some cases, it is more commonly met with in association with the staphylococcus and the diplococcus pneumoniæ. (3) *Tuberculous meningitis*, the variety characterised by the presence of the bacillus tuberculosis. (4) *Meningococcus meningitis*, the form distinguished by the presence of meningococcus or diplococcus intracellularis. This is better known as cerebrospinal fever or epidemic cerebrospinal meningitis. A modification of this organism is found in the posterior basic meningitis. From this it is obvious that the source of infection is in tuberculous, suppurative, or the host of other infective conditions affecting the body or the more closely related cranial accessory cavities and air sinuses. In all cases, therefore, of meningitis a careful examination should be made, not only with a view to ascertain the existence of a general pyæmic condition, but also the presence of disease of the middle ear, the nose, the frontal sinuses, the orbits, the naso-pharynx, as well as the possibility of fracture of the cranial bones (see Vol. I. p. 182).

From the numerous facts which are coming to light upon the causation and bacterial nature of acute meningitis, a classification according to present conceptions is a matter of considerable difficulty, but the following table, modified from the recent work of Dr. Osler (*The Etiology and Diagnosis of Cerebrospinal Fever*. London, 1899), would seem to embrace most varieties of the acute disease.

1. Primary meningitis:—
   - (*a*) Cerebrospinal fever—diplococcus intracellularis.
   - (*b*) Posterior basic meningitis—diplococcus intracellularis (modified).
   - (*c*) Pneumococcus meningitis—diplococcus pneumoniæ.
2. Secondary meningitis:—
   - (*a*) Tuberculous meningitis—bacillus tuberculosis.
   - (*b*) Pneumococcus meningitis secondary to pneumonia, endocarditis, and disease or injury of the cranial bones and accessory cavities.

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(*c*) Pyogenic meningitis—streptococcus and staphylococcus.
(*d*) Miscellaneous forms due to the bacillus typhosus, bacillus influenzæ, gonococcus, bacillus diphtheriæ, etc.

In the ensuing description there will be given first the general symptoms which indicate the presence of an acute inflammatory condition of the meninges, with an account of the differential diagnosis and treatment, and then there will follow a brief clinical account of the several varieties of leptomeningitis. This method has been adopted, as the main phenomena of acute meningitis remain fairly constant, whatever be the source of infection.

The **symptoms** are usually grouped into those occurring at different periods of the disease, such as the premonitory, the irritative, and the paralytic phenomena. PRODROMATA, mostly in the form of general malaise, headache, vomiting, and irritability, may exist in a number of instances; but their presence is by no means constant, the IRRITATIVE FEATURES of the disease often being the first indication, and coming on with extreme suddenness. Of these by far the most characteristic are ***headache***, usually of great severity, persistent in character, and located chiefly over the frontal region, but not limited to this or any other particular area of the skull. Though more or less constant from the outset until the paralytic stage is reached there are accessions of greater severity, so acute as to be productive of a cry, the so-called "hydrocephalic cry."

The co-existence of headache and delirium is generally regarded as characteristic of meningitis; in general febrile states with cerebral symptoms the headache ceases when the delirium begins.

Vomiting of the cerebral type is an early, a frequent, and often a persistent symptom.

The *delirium* is characterised by wandering of the mind and muttering, incoherent talking, sometimes associated with great activity and violence. Having set in it usually persists, the patient falling into a state of stupor, which eventually passes into coma, in which death occurs.

The *temperature* is variable; in cases presenting no exceptionally acute phenomena an elevation of two or three degrees is common. In acute septic conditions the temperature may rapidly rise to hyperpyrexia. In the cerebrospinal form a chart resembling that of typhoid fever is not uncommon; in the tuberculous variety a moderate elevation to 100° or 101° F. with a fall to normal or even below normal is observed.

Various *local phenomena* of an irritative or convulsive nature

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indicate more or less precisely the part of the brain chiefly implicated. Retraction of the head, and rigidity of the muscles of the neck and back, often to a marked extent, is common when the meningitis involves the base posteriorly; should it be situated further forward in the region of the optic chiasma and interpeduncular space, squinting, sometimes of irritative, sometimes of paralytic nature is evident, also ptosis, and in more chronic cases blindness may be observed. The other cranial nerves are more rarely involved. Should the meningitis implicate the convexity chiefly, then local or, more frequent in children, general convulsions are detected. The type of the convulsion depends upon the particular cortical area primarily involved, as elsewhere explained (p. 119). In place of convulsion there may be rigidity or even one-sided paralyses, but these are rare.

Optic neuritis may occur in acute meningitis, but it is not common, and when it does occur it is only in the later stages.

A sign to which attention has recently been drawn, but which is not characteristic of any special form of meningitis, is known as *Kernig's sign*. To obtain this the patient is placed in the dorsal decubitus, the legs being relaxed and extended at the knees. If now raised to a sitting posture, the knees are found to become flexed and cannot be straightened. When again laid on the back, it is found impossible to straighten the knees. This is due to strong contraction of the hamstring muscles.

The stage of irritation characterised by the symptoms just detailed passes eventually into the third or PARALYTIC STAGE. The delirium of the earlier days becomes stupor, and later on coma. The abdomen is markedly retracted, the pupils dilated, and loss of control over the bladder and rectum ensues.

The *duration* of a case of meningitis varies largely according to the nature of the infective process. The most rapid, as well as the most severe, is the acute septic or streptococcus meningitis, where the disease lasts a few days to two or three weeks. In the tuberculous variety the process is much more insidious, and the course more prolonged, lasting from six weeks to two months. In cerebro-spinal fever the duration is roughly estimated at from one to six weeks in cases of average severity.

Diagnosis.—The diagnosis of meningitis is often a matter of extreme difficulty, for, on the one hand, the onset of the affection in children resembles the incidence of an ordinary acute inflammatory condition, such as a febrile attack, an exanthem, or pneumonia, while, on the other hand, it may be grafted upon an already existing

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disease, such as pneumonia, suppurative otitis media, or a local tuberculosis.

In persons about the period of puberty, and in young adults, the difficulties met with in children rarely arise; but fresh perplexities confront the physician, the chief of which lie in differentiating the meningitis—more especially the tuberculous variety—from certain general diseases of a pyrexial nature, the most important of which is enteric fever. It is in the distinguishing of these conditions that some of the most difficult problems in clinical medicine arise. The four principal affections with which the disease may be confounded are typhoid fever, pyæmia, acute general tuberculosis, and apical pneumonia.

1. *Typhoid fever.*—In the examples of this disease in which there is no rash, or in which the rash is late in appearing, where constipation is a prominent symptom, where marked and persistent irritability exists, and in which the temperature remains between 101° and 103° F. for some weeks, the diagnosis may be impossible. The points to which attention should be directed are, a dorsal decubitus in typhoid, a lateral in meningitis. In the latter there is usually some retraction of the head and neck and continuance of headache even after the appearance of delirium, a retracted abdomen, a tendency to vomiting, with stupor eventually becoming coma. A greater irregularity of pulse, as a rule, characterises meningitis, more especially the tuberculous variety. In all doubtful cases the Widal test should be applied (Vol. I. p. 102).

2. *Pyæmia*, manifested by thrombosis of the cerebral venous sinuses (cavernous chiefly). The difficulty in diagnosis here lies in the fact that the local symptoms indicating thrombosis—protrusion of the eyeball, chemosis and conjunctival œdema, hæmorrhagic retinitis, and optic neuritis—do not immediately reveal themselves. A source of infection is, however, more likely to be obtained in these cases, the chief being tonsillitis of an influenzal or diphtheritic nature.

3. *Acute tuberculosis.*—As almost all the phenomena of enteric fever may be present in this condition, the same difficulty exists in distinguishing it from meningitis. The abdomen is usually retracted, the temperature variable, the pulse softer, not infrequently cough, while patches of broncho-pneumonia may possibly be detected on auscultation.

4. *Apical pneumonia.*—In the early stages of this condition the symptoms are largely, if not entirely, cerebral. The initial headache may be severe and the temperature high, but with the onset of

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delirium the headache as a rule subsides. The presence of tubular breathing at a lung apex will establish the diagnosis, although in some cases it may be late in making its appearance.

Lumbar punoture has been used both as a diagnostic and as a therapeutic procedure. It consists in passing a hypodermic needle between the laminæ of the third and fourth lumbar vertebræ into the sub-dural space and withdrawing some of the cerebrospinal fluid. An examination of the fluid thus abstracted has been of assistance in confirming or otherwise influencing the diagnosis of the several forms of meningitis. Positive evidence, in the presence of micrococci, or of pus, would appear to be of value, but negative results should be viewed with distrust. Tubercle bacilli have not invariably been found in cases of tuberculous meningitis, but the existence of pneumococci, staphylococci and streptococci point to the presence of a suppurative meningitis. The method has been largely used in the diagnosis of cerebrospinal fever, where the presence of pus, with or without the co-existence of the characteristic meningococcus, has been of diagnostic service. It is undoubtedly of value in showing the presence of an excess of cerebrospinal fluid.

As a therapeutic agent, lumbar puncture has not taken the place which its advocates originally expected of it. A temporary relief may follow the withdrawal of a small quantity of fluid in cases of suppurative meningitis, but its use in cases of intracranial, and more especially cerebellar tumour, with a view to the relief of excessive pressure, has in many cases been distinctly harmful.

Treatment.—Meningitis being an acute infective disease, as already shown, the most important feature in the preventive treatment lies in careful attention to, and active surgical interference with, pyogenic disorders of the cranial accessory cavities, more especially the ears, nose, mouth, and orbits. As it may also be secondary to general morbid conditions, such as pneumonia, endocarditis, pyæmia, and influenza, the early diagnosis of such affections is highly desirable; and it is scarcely necessary to do more than mention the necessity of careful antiseptic treatment of all scalp wounds, and injuries of the cranial bones.

The diagnosis of meningitis having been established, either from the clinical phenomena alone, the co-existence of aural or other adjacent suppuration, or from bacteriological examination by the aid of lumbar puncture, the treatment lies merely in the carrying out of certain general principles. The head should be shaved and ice-bags applied; cupping may be advantageously effected, and even venesection in suitable cases.

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The administration of drugs to overcome the pain and allay the spasmodic and convulsive affections is desirable. For this purpose the bromides, either alone or in association with chloral, may be given by the mouth; or chloroform may be used as an inhalation. In other cases the hypodermic injection of morphia may be necessary. The constipation, usually so marked, requires attention.

The question of surgical interference may arise. In every case it is assumed that attention has been paid to the accessory cavities; careful washing out and syringing will not bring about such effective results as opening the mastoid or frontal sinuses, should these be the primary source of infection. Opening the subdural space is not likely to be of any value, as the extension of inflammatory products along the blood-vessels and downwards towards the spinal cord is rapid, and it is naturally impossible to wash out the pia-arachnoid spaces in which the materies morbi is chiefly located. The uselessness of lumbar drainage in this condition has been mentioned when that procedure was described.

Further reference may be made to the handling of the special forms of meningitis. As recovery is not unknown in *cerebrospinal fever*, the chief treatment lies in the administration of stimulants and nutritious food, and the combating of special symptoms—pyrexia, etc.—as they arise. In *tuberculous meningitis* many remedies have from time to time been recommended — mercury, the iodides, iodoform, guaiacol, tuberculin, etc. The disease is almost uniformly fatal, and little can be done after the initial symptoms have arisen. In prophylaxis lies the only satisfactory agent.

Following upon the description, just given, of acute lepto-meningitis, there is now added a short account of the varieties of this disease—variations due to either special clinical phenomena, the age at which they occur, their course, or the infecting organism.

1. POSTERIOR BASIC MENINGITIS

This is a condition occurring chiefly in infants under one year of age. Of 110 cases described by Dr. Lees and Sir T. Barlow, 84 were observed during the first year of life. The onset of the disease is usually sudden, and it may not be possible to connect it with any infective causation. The usual causes of meningitis, as a rule, are absent, such as otorrhœa, congenital syphilis or tuberculous disease. The malady is characterised by a marked and persistent retraction of the head and by vomiting; sometimes by convulsion.

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This is more commonly of a tonic than of a clonic nature, affecting the deep cervical muscles, the limbs, and the trunk, to such an extent that in some opisthotonos is well marked.

Various ocular symptoms may be superadded to the above: these are retraction of the eyelids, strabismus, occasionally jerking movements of the eyeballs, and in a few instances blindness.

As a rule stupor ensues and deepens into coma, in which the patient dies, at a period varying from one or two weeks to three or even four months after the onset of the symptoms.

The characteristic pathological appearances consist of a purulent or fibrino-purulent effusion over the posterior basic region, involving more especially the fold of arachnoid membrane which passes from the under surface of the cerebellum to the medulla oblongata. From this it may extend downwards over the spinal cord, or upwards into the ventricles; more rarely it may pass forward to the optic chiasma and interpeduncular space. Owing to the effusion in the posterior basic region the foramina of Key and Retzius and Luschka became blocked, and the condition known as acquired hydrocephalus ensues.

How far this condition is to be regarded as separate, or, as some suggest, a modified and sporadic form of cerebrospinal meningitis, can only be decided by bacteriological examination. The observations of Dr. Still have shown the presence of a diplococcus in the exudation of this disease, differing in some major respects from the D. pneumoniæ, and in some minor ways from the D. intracellularis of Weichselbaum. (For further details see Vol. I. p. 189.)

2. TUBERCULOUS MENINGITIS

Although most commonly seen in children between the ages of one and six years, the disease is by no means limited to them, as it occurs not infrequently in adults. Tuberculosis being an acquired disease, the presence of the tubercle bacillus is essential for its development. In the case of the cerebral meninges, infection is usually secondary to a tuberculous focus in the brain or other part of the body, such as the lungs and pleura, the joints and bones, the testicles or the lymphatic glands. Tuberculous meningitis is undoubtedly facilitated by conditions which lower the resistance of the tissues: such are, improper or insufficient food and bad hygienic surroundings, overcrowding, and want of fresh air.

The **pathological appearances** of tuberculous meningitis are sufficiently characteristic. Over the base, more especially in the

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region of the optic chiasma and interpeduncular space, the pia-arachnoid is thickened, opaque, and of a matted appearance. Here also is found an exudation, chiefly of a fibrino-purulent character, which extends along the fissure of Sylvius towards the convexity, and posteriorly over the sides of the pons Varolii. The exudation, however, is not limited to these parts, as it may be found on the fold of pia-arachnoid between the cerebellum and the medulla oblongata, and on the superior surface of the middle lobe. The ventricles are usually distended, and may at times contain a semipurulent fluid. The structures adjacent to the ventricles are commonly found after death to be in a condition of extreme softening, sometimes amounting to a creamy consistence. Some maceration of the tissues may occur during life, but it is chiefly a post-mortem change. Besides the exudation, tubercles varying in size, but usually not larger than a pin's head, are observed along the lines of the blood-vessels. In many cases a mixed infection is present when strepto- and staphylococci are associated with purulent effusion.

Symptoms. — The symptoms of tuberculous meningitis are those of ordinary acute leptomeningitis already described, modified by the seat of the lesion, which is usually median basic, and by the subacute nature of the specific inflammatory process.

The onset of the disease is characterised by various gastro-intestinal symptoms, such as nausea, vomiting, diarrhœa, or constipation, and evident malaise.

Following upon these are symptoms indicating irritation of the structures of the base of the brain, the cortex or other locality, which may be affected. Hence there may be observed general or local convulsions, paralysis of the ocular or other cranial nerves, retraction of the head and neck, and arching of the back.

These later on give way to paralytic phenomena: such as paralysis of a hemiplegic or monoplegic character, ptosis, mental hebetude, and apathy; later on stupor passes into coma, the breathing assumes the Cheyne-Stokes type, urine and fæces are passed unconsciously, and death eventually ensues.

The *temperature* in tuberculous meningitis presents irregularity as its essentially characteristic feature. There is often an initial fall; in the later stages the temperature rises, sometimes to hyperpyrexia.

The *pupils* are contracted in the early stages; with increase of the intracranial pressure they are observed to dilate, and in the later periods the light reflex may be abolished.

The *pulse* is characteristically irregular both in frequency and

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volume; an initial acceleration may be succeeded by a slowing and an irregularity of the beats with increase of tension. Towards the end the rapidity of the beats is excessive. Tubercles are occasionally seen in the choroid.

3. CEREBROSPINAL FEVER

The features of this condition, by which its presence may be suspected, and eventually differentiated, from other forms of meningitis, are:—First, its occurrence usually in epidemic form; secondly, its tendency to attack children as well as young adults to an almost equal extent; and thirdly, the abrupt onset of the symptoms, with severe occipital headache, vomiting, and pyrexia. The course of the disease varies greatly in different cases. According to Dr. Osler the following types are met with. In some the temperature varies little from an average case of typhoid fever; others present the remissions commonly associated with tuberculous disease; in others, again, the disease is characterised by morning pyrexia; while in not a few cases the chart is suggestive of malaria.

Amongst the more important and characteristic symptoms of the affection are various skin rashes of an erythematous and herpetic nature, while in the malignant forms purpuric blotches are seen. Arthritis and periarthritis are also more common in this than in other forms of meningitis.

For fuller account see Vol. I. p. 182.

4. PNEUMOCOCCUS MENINGITIS, PYOGENIC MENINGITIS, MENINGITIS OF MIXED INFECTION

These forms of meningitis are classed together, as it is not yet clear how far they may be clinically separated from each other. The diplococcus pneumoniæ has frequently been found in fatal cases of meningitis, both alone and in association with the pyogenetic organisms, the strepto- and staphylococcus. A primary pneumococcus meningitis is described, but more commonly the infection is secondary, and associated with general pyæmic conditions, such as empyema and ulcerative endocarditis; while a not infrequent source of infection is to be found in local conditions in the ear, nose, mouth, and orbit.

The symptoms are those of acute leptomeningitis, of which it is the most severe variety, running a rapid course, and ending fatally in coma. The onset of the symptoms may be obscured by the

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disease, of which the meningitis is an intercurrent affection. (See Suppurative Meningitis, Vol. I. p. 196.)

The annexed Table shows the chief characteristics of the several forms of meningitis described in the text :—

	1. Posterior Basic.	2. Tuberculous (median basic).
(*a*) Age	Under 1 year	Children and adults under 50.
(*b*) Course	A week to 3 or 4 months	6 weeks to 2 months.
(*c*) Organism	A special diplococcus	The tubercle bacillus.
(*d*) Infection	Primary; sporadic	Secondary to tubercle elsewhere.
(*e*) Prognosis	Commonly fatal	Fatal.
(*f*) Special feature	Marked and persistent head retraction	Irregular temperature and pulse; frequent strabismus; marked constipation.

	3. Cerebrospinal Fever.	4. Mixed Infection (chiefly pneumococcus meningitis).
(*a*) Age	Children and young adults	All ages.
(*b*) Course	Onset abrupt; duration 1 to 6 weeks	A few days to 1 week.
(*c*) Organism	Diplococcus intracellularis	Pneumococcus, streptococcus, staphylococcus.
(*d*) Infection	Primary; epidemic	Always secondary.
(*e*) Prognosis	Recovery not uncommon	Invariably fatal.
(*f*) Special feature	Erythematous and herpetic rashes	

Hydrocephalus

The word hydrocephalus has been used to designate two different conditions, termed respectively the acute and the chronic. *Acute hydrocephalus* has been applied to that variety of tuberculous meningitis in which there is marked ventricular (internal) or subpial (external) sero-purulent effusion. The term, however, is a misnomer, and confusion has arisen over its use; it is best to limit it to the chronic variety.

Chronic hydrocephalus.—This is a disease of infancy, arising from an excessive accumulation of cerebrospinal fluid within the ventricles of the brain. It is usually congenital; more rarely acquired. It is due to an obliteration, inflammatory or developmental, of the foramen of Magendie, of the foramina of Key and Retzius, and of the iter Sylvii. Resulting from the excessive internal

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pressure in the ventricles, there is a thinning of the white matter, and a flattening of the cerebral convolutions, a separation of the bones of the skull, opening up of the cranial fontanelles and sutures, and enlargement, sometimes to an enormous extent, of the head.

It may be predisposed to by parental syphilis and alcoholism, and developed by the onset of rickets.

Symptoms.—The head begins to enlarge shortly after birth, or the infant may be born with so large a head that instrumental interference is required. As the child grows the head increases out of all proportion to the face and body; there is a want of mental development and intelligence, and fits may ensue. If the case is slow, walking is retarded. The skull is usually elliptic in shape, the vault elevated and convex, the sutures open, and the superficial veins distended. The hydrocephalic skull presents a distinct contrast to the big head of rickets, with the thickened and prominent frontal and parietal eminences and flattened vertex, not infrequently asymmetrical in shape. Sight and hearing are frequently retained, but squinting and optic atrophy are not unusual.

The disease may run a short course, the child dying in a few months, or at one or two years of age. Many, however, live longer, but die before puberty; some may reach adult age.

Treatment.—To be of any material value the treatment should consist of removal of the intra-ventricular fluid in the early stages. A method has been recommended of draining the ventricles through an opening in the cortex cerebri. Mere tapping the ventricles is of little use, as the fluid rapidly reaccumulates. Lumbar drainage is of no value should the foramen of Magendie be obliterated.

Sinus Thrombosis

Clotting of the blood in the intracranial sinuses is a serious and often a fatal condition. The infective variety is by far the more important; but a primary or marasmic form has been described.

1. *Marasmic.*—In the latter form the thrombus tends towards organisation and absorption, and general symptoms are rarely present. The condition is uncomplicated, and occurs chiefly in the superior longitudinal sinus. It is found, for the most part, in infants weakened by prolonged diarrhœa; but all debilitating diseases predispose towards it, more especially those interfering with respiration and the action of the heart. In adults it is found more commonly in chlorosis, in the late stages of phthisis, in typhoid fever, in parturition, and in the marasmus of cancer.

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The **symptoms** are those of obstruction of the superior longitudinal sinus. As a result there is distension of the nasal veins and epistaxis; and of the veins generally over the scalp. Various phenomena referable to disturbance of the cerebral circulation are also seen; such are, convulsions and paralysis, vomiting and headache, somnolence, delirium, stupor and coma.

2. *Infective.*—Sinus thrombosis of an infective or inflammatory nature is secondary to infective conditions in other localities. It is a disease more commonly seen in adults, rarely in old people and in children. The prevailing cause of intracranial thrombosis is a chronic suppurative inflammation of the middle ear, with a carionecrotic affection of the surrounding bone; but inflammatory conditions of other accessory cranial cavities, and fracture of the skull involving the diplöe, are rarer but important causes. But other processes should not be forgotten in this connection: such are erysipelas of the face and scalp, dental caries with periostitis, faucial inflammation, and retropharyngeal abscess.

The **symptoms** are indicative of a general pyæmic poisoning of the whole system, as well as those referable to implication of particular sinuses. Hence there fall to be described the general and the local phenomena.

The *general symptoms* are headache and vomiting, the former being severe and usually localised to the neighbourhood of the primary lesion; the temperature is of a characteristically remittent type, with sudden elevations to 104° or 105° F., associated with a small, rapid, and irregular pulse, rigors, vomiting, and profuse sweating. In unfavourable cases there follow indications of interference with the functions of the lungs and the abdominal organs. By this means Macewen has differentiated two types of the pyæmic infection —the pulmonary type, which shows itself by cough, pain in the chest, "prune-juice" expectoration, and fetor of breath—symptoms which indicate pulmonary infarction and pneumonia; and an abdominal type characterised by a dry, cracked tongue, vomiting, tympanites, diarrhœa, and the general features of the typhoid state.

Meningitis may ensue upon a sinus thrombosis, either as a result of the causal inflammation, or by direct extension from the thrombosed venous sinus. In this event, to the already mentioned symptoms are added irritability and restlessness, tonic and clonic spasms of the face, neck, and limbs, and later on delirium and coma, in which death ensues.

The local symptoms point to two chief varieties of sinus thrombosis:—

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(1) Of the *cavernous sinus.* This is secondary to infective conditions of the orbit, face, mouth, nose, throat, and pharynx. The chief localising features are chemosis, or œdematous swelling of the conjunctiva, proptosis, or protrusion of the eyeball, and œdema of the upper eyelid and root of the nose. Then ensue interference with the ocular movements, either directly from œdema and venous engorgement, or from paralysis of the third nerve, while later on optic neuritis may develop.

(2) Of the *lateral sinus.* This is secondary to chronic purulent inflammation of the middle ear with bony cario-necrosis; to diphtheritic or scarlatiniform inflammation of the fauces; to infected wounds of the head, neck, and mastoid region, and to fracture of the base of the skull.

Clinically, the local appearances are distension of the veins over the mastoid region, œdema over the mastoid process, and hardness of, with pain on pressure over, the internal jugular vein and in the upper part of the posterior triangle of the neck. There is also pain in, and the presence of an offensive purulent discharge from, the affected ear. Optic neuritis may be present in lateral sinus thrombosis; if so it indicates an extension forwards of meningitis, and is an urgent symptom.

Pathology. — The morbid appearances of a cranial sinus thrombosis resemble those of infective phlebitis in other localities. There is a collection of pus between the cranial bones and the dura mater, forming a small subdural abscess. As a result of this adjacent infection, the lateral sinus lying between the two layers of the membrane becomes filled with a clot in various stages of disintegration; and the vessel walls are similarly affected. In the clot are the micro-organisms commonly associated with purulent collections, the strepto- and staphylococci. From this region the general vascular system becomes secondarily infected, and the symptoms of pyæmia are therefore early apparent. In a like fashion an infective thrombus acts as a source of local infection, and may give rise to purulent meningitis and intracranial abscess.

Diagnosis.—Although this condition may be recognised from the symptoms already narrated, some points may require special emphasis. In some cases pain in the ear and otorrhœa may be entirely absent, the aural discharge having stopped some time before the onset of the symptoms. The disease with which it is most likely to be confused is typhoid fever, more especially if this is complicated with chronic otorrhœa. Exact diagnosis may be almost impossible, if lateral sinus thrombosis is complicated with

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cerebral abscess and suppurative meningitis. The points by which an uncomplicated case may be differentiated from tuberculous meningitis and cerebral abscess will be detailed in the Table on p. 90.

The **prognosis** of infective thrombosis is unfavourable. If surgical interference is early and thoroughly performed, local or general infection may be prevented, but the diagnosis is rarely possible before the general symptoms make their appearance. The duration of an untreated case of infective sinus thrombosis is approximately three weeks (Ballance).

The **treatment** is entirely surgical, and limited to affections of the lateral sinus. After exposure the occluded portion of vein is ligatured and excised. As cerebral or cerebellar abscess may co-exist with sinus thrombosis, attention should be directed both to the temporo-sphenoidal lobe and the cerebellum.

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CEREBRAL VASCULAR LESIONS

Two conditions usually mentioned in the text-books require a brief reference, mainly because important symptoms have been ascribed to their influence. They are *Cerebral Hyperæmia* and *Cerebral Anæmia.* The physiology of the cerebral circulation, however, would appear to show that little variation takes place in the total quantity of blood at any one time within the cranium (Hill). Cerebral arterial anæmia is occasioned physiologically by congestion of the venous circulation, as occurs in dilatation of the right side of the heart, and in shock and collapse, and temporarily in conditions of straining, such as holding the breath, coughing, and in epileptic convulsions. It may also be brought about from dilatation of the arterioles, which occurs around tumours and patches of inflammation. It also occurs as part of the chlorotic state in girls, and in severe anæmias of whatever nature. The only condition which would produce general cerebral hyperæmia is an acceleration of the venous circulation and absorption of the cerebrospinal fluid. The influence of general cerebral hyperæmia and anæmia, as factors in disease, is therefore doubtful; and it is highly improbable that the protean symptoms of neurasthenia are solely due to any active or passive congestion of the brain, as stated by some authorities.

Local cerebral anæmia—ischæmia—is due to temporary obstruction in, or obliteration of, a cerebral arteriole, the results of endarteritis. The symptoms of this condition are well known, and are described in the account of thrombosis and cerebral syphilitic affections.

The more important vascular lesions which will be here described are those resulting from rupture of a cerebral artery—hæmorrhage, or blocking—embolism and thrombosis; but before describing these affections it is necessary to refer to the clinical features of apoplexy, or the comatose state.

Apoplexy

This is a condition, the onset of which is characterised by sudden unconsciousness, and is commonly due to rupture of a cerebral blood-vessel.

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The clinical features of a case of apoplexy will first be described. The method of onset varies, but is always sudden. Common variations in the mode of commencement are:—(1) Sudden coma, lasting for a longer or shorter period, in which the patient may die, or from which recovery may take place with a resulting hemiplegia. (2) Convulsions and coma—the so-called epileptiform apoplexy.

Should a history be obtained, it will in some cases be found that, although the coma came on suddenly, it had been preceded for a few hours, or perhaps for a day or two, by giddiness, a feeling of fulness in the head, or numbness or tingling in an arm or leg. But in many cases coma is actually the first symptom.

The aspect of a patient with apoplexy is characteristic. There is unconsciousness, stertorous breathing, and a cyanosed appearance of the face. If the coma is profound, it is difficult to detect which side is paralysed, but rigidity of one side, and flaccidity of the other, may be observed. The *temperature* is at first depressed a degree or two, to be afterwards followed by a rise. A steady elevation to considerable pyrexia is ominous. In cases likely to recover the temperature does not rise beyond 101° F. or 103° F. at the highest. That of the paralysed is about a degree higher than on the non-paralysed side. The *respiration* is "puffing" or stertorous, and in fatal cases early assumes the Cheyne-Stokes type. The *pulse* is tense and slow, but later may become increased in frequency. The *head* and *eyes* are often deviated; in the ordinary capsular hæmorrhage the deviation is away from the palsied limbs; the patient, it is said, "looks to the lesion." The pupils may be small or dilated; if unequal, the larger is usually on the side of the lesion. The *knee-jerk* is often lost on the paralysed side.

In the so-called epileptiform apoplexy, the patient may be found in a state of coma, with recurring attacks of convulsions affecting both, or, more commonly, one side of the body, which side, should recovery take place, will be found paralysed.

Apoplexy may occur from hæmorrhage into various parts of the brain, and it is therefore necessary to consider by what features, if any, bleeding into several common localities may be distinguished.

(1) *Capsular hæmorrhage*, or effusion of blood into the internal capsule, basal ganglia, and adjacent tissues.—This is the common form, and the one which gives the type to the account of apoplexy. It is characterised by coma and the other phenomena already described.

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(2) *Subdural hæmorrhage.*—Occurring sometimes in infancy, and from traumatic causes, it is usually found in adults in chronic alcoholism and in general paralysis of the insane. It is distinguished by unilateral convulsions ending in paralysis and coma. If there is much compression of the brain, the pupil on the side of the lesion dilates. This is the lesion in pachymeningitis hæmorrhagica, which has been already described on p. 52.

(3) *Ventricular hæmorrhage.*—This may be primary, but is more usually secondary to rupture into the ventricle of a capsular hæmorrhage, and is then indicated by a marked deepening of existing coma, by rigidity giving place to flaccidity of the limbs on both sides, and by rapid death.

(4) *Pontine hæmorrhage.*—If the effusion be large, coma is profound, and accompanied by jerking movements of all the limbs; the pupils are contracted to pin-points, and the temperature rises to hyperpyrexia. The respiration may stop suddenly for some minutes before the heart ceases to beat, and death rapidly ensues. In localised or small pontine hæmorrhages the symptoms vary according to the locality, and are described later on in the account of focal diagnosis (p. 134).

(5) *Cerebellar hæmorrhage* is rare and difficult to detect. It may be preceded by headache and giddiness, or sudden coma comes on, terminating rapidly in death. If less severe there is vomiting and unsteadiness of gait.

The course of an apoplexy.—This has been incidentally referred to when describing the several varieties. The following types will include most of the cases met with in practice:—

(1) Death within a few hours without return to consciousness; and in some cases of pontine hæmorrhage within an hour of the onset.

(2) Partial recovery, but death occurring from the second to the fourth day after onset, during the period of so-called "reaction."

(3) Recovery from the initial phenomena, but either as a result of renewal of the hæmorrhage, from inflammatory action at the site of the bleeding, or from pulmonary complications, death results between the second and the fourth week.

(4) Recovery is maintained over the fourth week, and the chronic stage of hemiplegia is apparent.

Differential diagnosis of the comatose state.—In every case of coma the practitioner should (*a*) obtain a sample of the urine (by catherisation) and test for albumen and sugar; (*b*) take the temperature of the body; (*c*) note the state of the respiration,

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pulse, pupils, and the knee-jerks; (*d*) examine the skull, and (*e*) when possible obtain information as to the nature of the onset.

The following possible causes of coma should be borne in mind:—

Alcoholic intoxication.—It is often impossible to distinguish the unconsciousness of an alcoholic bout from that of cerebral hemorrhage or fracture of the base of the skull. The following evidence is strongly in favour of alcoholic poisoning—A marked alcoholic odour of the breath, the presence of alcohol in the stomach washings and in the urine,[1] unassociated with the signs of fracture of the skull, such as bleeding from the ears, nose, or mouth, of albuminuria and glycosuria, and of unilateral paralysis. But cases are frequently seen, more especially in hospital practice, where there exists a combination of factors inducing the comatose state; such are alcoholic intoxication, with fracture of the base of the skull, or with cerebral hæmorrhage, or with cerebral hæmorrhage and uræmia, associations which present an almost insurmountable difficulty of diagnosis.

Uræmia.—The coma of uræmia is less profound than that of cerebral hæmorrhage; the patient may be temporarily roused, and the onset is more gradual; the urine is albuminous, and the quantity of urea is diminished. But the presence of albuminuria in a comatose person does not necessarily imply uræmic coma. The co-existence of albuminuric retinitis strongly favours the diagnosis of a uræmic condition.

Narcotic poisoning.—Here the onset of coma is slow, the pupils contracted, the respirations infrequent, and the skin moist.

Diabetes mellitus.—The presence of a large quantity of sugar in the urine and the characteristic odour of the breath are usually sufficient to establish the diagnosis.

Post-epileptic coma.—This is of short duration, the knee-jerks are exaggerated, and on recovery the tongue is frequently found to have been bitten.

Cerebral hæmorrhage.—Coma may follow hæmorrhage into any part of the brain. If large and into the ventricles the coma may be profound, or there may co-exist rigidity of the limbs on one or both sides. The temperature may at the outset be subnormal, and Cheyne-Stokes respiration may ensue. The presence of albuminuria with coma in an elderly person is highly suggestive of cerebral hæmorrhage.

[1] If a few drops of urine be added to a solution of pot. bichrom. 1 part, and acid. sulphur. fort. 300 parts, and a bright emerald green coloration results, strong evidence of the presence of alcohol is obtained.

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Fracture of the skull.—In most cases there is external evidence of injury or bleeding from the ears, nose, or mouth. The coma is almost invariably due to the presence of hæmorrhage from rupture of the meningeal or other blood-vessels.

A few rarer causes of coma may also be mentioned: such are intracranial tumour and abscess, general paralysis of the insane, heat exhaustion and sunstroke; while a state of stupor may be seen associated with catalepsy and cerebral syphilis.

HÆMORRHAGE

Cerebral hæmorrhage is a disease of adult life, the greatest number of cases occurring during the fourth and fifth decades. But statistics also show that there is a special liability to its occurrence during the first year of life; while it is relatively infrequent in old age.

Hæmorrhage is one of the causes of "stroke," and, as already mentioned when apoplexy was under consideration, may occur into any part of the brain. The relative frequency with which different regions are attacked is as follows:—(1) The internal capsule, basal ganglia, and neighbourhood. Here hæmorrhage commonly is due to arterial disease; more rarely to traumatic causes, as is sometimes seen in severe concussion and in fracture of the skull. The vessels usually implicated are the lenticulo-optic and the lenticulo-striate branches of the middle cerebral artery, which supply the basal ganglia and internal capsule. (2) The meninges, where the cause is chiefly traumatic, but may be spontaneous, as in hæmorrhagic pachymeningitis. (3) The cerebellum. (4) The pons, optic thalamus, and quadrigeminal region. In these atheroma is the underlying pathological condition.

Cerebral hæmorrhages may be multiple, by which is meant either a number of small hæmorrhages occurring at or about the same time in different localities, or a single hæmorrhage occurring at successive periods. These are seen usually in states of general vascular degeneration, such as is met with in senility, and atheroma of the cerebral arteries with or without renal cirrhosis. They are not infrequent in the pons Varolii, where, if symmetrical and bilateral, a variety of "bulbar palsy"—pseudo-bulbar paralysis—is clinically apparent.

The pathological conditions favouring rupture of the cerebral arterioles are—(1) Degeneration of the coats of the small arteries, giving rise to the production of miliary aneurysms. This may occur

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during the degenerative period of life, quite apart from the co-existence of chronic renal, or cardiac disease.

(2) A degenerative condition of the walls of the arterioles incidental to chronic renal cirrhosis and hypertrophy of the heart.

(3) Vascular degeneration occasioned by chronic alcoholism and gout.

Under this heading may also be included those general diseases which give rise to bleeding; such are purpura, leucocythæmia, and pernicious anæmia.

(4) Cerebral hæmorrhage may arise from syphilitic disease of the arteries, but is much less frequent than thrombosis.

A hæmorrhage having taken place into the central parts of the brain insufficient to cause death, certain reparative changes occur in the clot, and degenerative alterations in the nerve tissues. The former consist in coagulation and absorption of the clot, the formation of a fibrous wall and cyst containing a serous fluid; the latter of degeneration of the neuronic systems whose nerve fibres have been destroyed. As a result of this various clinical phenomena arise, which are described under hemiplegia. The neuronic systems of the several cortical areas are referred to in the chapter upon localisation.

The **symptoms** of cerebral hæmorrhage vary somewhat, according to the region of the brain primarily affected. The onset is sudden, and not necessarily preceded by any premonitory sensations. Loss of consciousness is commonly profound, but there are cases in which the patient may be partially roused, and even put out his tongue and answer questions. In cases which recover, the interference with consciousness clears away in a few hours, and the limbs on one side are found to be paralysed, and there may be defect of articulation or aphasia if the left side of the brain in the region of the third frontal gyrus is involved. In a few days the period of "reaction" sets in, and "early rigidity" of the paralysed limbs is detected, and the temperature may be elevated a degree or two. This condition is ascribed to inflammatory changes around the cerebral lesion.

THROMBOSIS

Another cause of "stroke" is thrombosis, or blocking of a cerebral artery, the result of which is to cause softening in the area of arterial distribution. In some cases, however, the obliteration of the lumen of the vessel is incomplete, a condition of local

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anæmia—ischæmia—being produced, the circulation returning to the part either through absorption of the thrombus or by aid of collateral circulation. As thrombosis only occurs in diseased conditions of the vessel walls, chiefly of the inner coat, premonitory symptoms are frequent, owing to the existence of obliterative endarteritis for some period before the actual obstruction takes place. The premonitory symptoms are—numbness of the limbs on one side, transient monoplegia or hemiplegia, temporary aphasia and impairment or loss of memory. Hence, also, the onset of the thrombotic stroke is prolonged over a few hours, is rarely attended by loss of consciousness, and is favoured by conditions of low arterial tension, such as occur during sleep, in marasmus, and in extreme old age, but there is commonly some blunting of the intellect, confusion of ideas, impairment of memory and restlessness at night.

The pathological states underlying cerebral thrombosis are—(1) Syphilitic disease, chiefly in the form of endarteritis obliterans. This, rather than hæmorrhage, is the common cause of hemiplegia in young adults who have acquired syphilis. It affects mainly the larger arteries: internal carotid, middle cerebral, vertebral, and basilar. (2) Atheroma, or patchy degeneration of the arterial walls—endarteritis deformans. It is a disease of the degenerative period of life, and may or may not be associated with chronic renal cirrhosis. It is commonly associated with the gouty constitution. (3) An obliterating endarteritis is seen in frequent relation with chronic inflammatory conditions of the brain, with tuberculous growths, gummata, and sarcomatous and gliomatous tumours. This also is more common in the larger arteries, such as the middle cerebral, basilar, vertebral, and posterior cerebral.

The anatomical effects of thrombosis are seen in the several varieties of softening, characterised as white, yellow, and red. In the later stages, owing to breaking-up and absorption of the nervous and neuroglial tissues, a cystic formation may be developed, or the whole softened area may be replaced by a fibrous cicatrix.

The influence of thrombosis upon the affected neuronic systems is similar to that following cerebral hæmorrhage, viz. degeneration and sclerosis, which are shown clinically by the phenomena of old-standing hemiplegia.

EMBOLISM

In this the thrombus is carried from a distance, and most frequently from the heart, either as the result of mitral stenosis or of an acute endocarditis, the common affection being especially the

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ulcerative or malignant variety. From the association between embolism and infective conditions, such as pyæmia, abscesses are formed in the brain at the seat of embolic infarction.

As regards **symptoms** the onset is sudden, and usually in the course of an acute infective disease. Premonitory symptoms are rare, unless they be previous attacks of a similar nature. Consciousness is not entirely lost, and the hemiplegia is complete and frequently associated with aphasia, from the more frequent plugging of the left middle cerebral artery, owing to its more direct continuation from the internal carotid.

The points of **differential diagnosis** of the comatose state have been already mentioned; but it is important to be able to distinguish early in the course of the disease the condition underlying the "stroke," which results from the three forms of vascular lesion just described. In the following table the chief points by which this may be done are indicated:—

Hæmorrhage.	Thrombosis.	Embolism.
Commonest between forty and sixty-five years	In the old, the syphilitic, and in those with vascular degeneration	Chiefly in young adults.
Renal disease frequent; syphilis rare	Syphilis frequent, also renal disease	Associated with rheumatism, endocarditis, and infective conditions.
Premonitions rare; if any, vertigo and fulness in head	Premonitions usual; numbness, transient aphasia, monoplegia	Premonitions rare.
Onset sudden and acute	Onset prolonged over a few hours	Onset sudden.
Coma profound and progressive	Coma rare	Coma, if present, transient.
Initial fall of temperature succeeded by rise	Temperature rarely affected.	
Pulse full, tense, and regular	Pulse feeble, irregular, and compressible.	

The **treatment** of the comatose state is to a large extent the treatment of the condition which has given rise to it. In all cases of coma brought to the hospital in which a doubt is expressed as to the cause, whether it be alcoholic apoplexy or fracture of the skull, the patient should be given the benefit and detained. Even uncomplicated alcoholic coma should not be lightly regarded, but treated as a serious condition.

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In the coma of *cerebral hæmorrhage* the patient is to be placed in bed in a horizontal posture, the head being slightly elevated. An ice-bag should be applied to the head, and mild counter-irritants, in the form of mustard and hot-water cloths, to the extremities. Let five grains of calomel, or a drop or two of croton oil, be placed on the back of the tongue with a view to securing a free action of the bowels. In rare cases only is blood-letting likely to be of value, the indications for this being a hard and tense pulse, and a cyanotic appearance of the face. Ligature of the carotid artery on the side of the lesion has been recommended, but its value is at the best doubtful.

If the case is clearly of syphilitic nature (either from the history or from external evidence), large doses of iodide of potassium and inunction of mercury ought to be started at once; in non-syphilitic cases at this stage the administration of the iodides is of little use.

Various minor points, but ones upon which the future progress of the case largely hangs, require the physician's attention. The stertorous breathing, due to falling back of the tongue and epiglottis, a distressing feature of the comatose state, may be relieved by turning the patient on his side. The state of the bladder should be early investigated, and the catheter used regularly as long as the coma exists. Frequent washing of the mouth with mild antiseptic lotions, and attention to the skin, especially in the place of greatest pressure, such as the lower part of the back, should not be neglected.

The patient's *diet* has also to be considered. If the coma lasts only a few hours, no nourishment is necessary; should it be prolonged small quantities of milk, beef-tea, and soups may be frequently administered, if necessary, per rectum. A rapid and irregular pulse of low tension requires the use of alcohol in small or frequently repeated doses; if the tension is high, careful administration of the nitrites may materially relieve the cardiac distress.

If the "stroke" is clearly due to *cerebral thrombosis*, the diagnosis of which should be made at the earliest available opportunity from the clinical features and history of the case already detailed, some modification in the treatment is required from that recommended for cerebral hæmorrhage. The chief cause of this condition being, in adults, syphilitic endarteritis, the early and strenuous administration of anti-syphilitic treatment needs no further commendation. In later life, degenerative vascular change, with or without co-existent renal disease, is the most common cause. As a rule there is no urgency in the condition, so that if

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the patient is kept entirely at rest in the recumbent posture, a light nutritious diet given, and alcoholic stimulants avoided, unless there exists cardiac failure, with usually the administration of bromide of ammonium, if there be restlessness at night, recovery from the immediate effects of the stroke will result.

In cerebral *embolism* the treatment is directed to the general condition which has given rise to the embolus. This is most commonly pyæmia with ulcerative endocarditis, the treatment of which is elsewhere described.

HEMIPLEGIA

Hemiplegia of sudden onset results from one or other of the three vascular lesions just described. If from hæmorrhage, it is due to laceration of the motor fibres; if from embolism or thrombosis, it is associated with softening of the cerebral tissues arising from impaired or arrested blood supply. Ingravescent hemiplegia, or gradually developing paralysis of one side, is characteristic of tumour formation within the skull.

For descriptive purposes hemiplegia may be divided into two stages:—(1) That observed during the first three or four weeks following the "stroke," characterised by more or less complete paralysis of one side with flaccidity of the muscles; and (2) that which arises from the fourth to the eighth week and continues, the features of which are partial paralysis, rigidity, and contracture.

The method of recovery of the paralysed members follows certain definite lines. The common observation is that the face recovers before the leg, and the leg before the arm. The coarser movements at the large joints recover before the finer movements at the small joints; the hand, however, rarely regains the power of precise movement which was present before the attack. But certain cases show variations from this. There are clinically observed: (*a*) The facio-lingual type, in which the face and tongue are as much as, or even more paralysed than, the limbs. The oculo-facial muscles are rarely as much affected as the oro-facial. If the hemiplegia is right-sided and of cortical nature, this variety is associated with motor aphasia. (*b*) The arm, or common type, in which the upper limb is most paralysed, the leg to a lesser extent, and the face and tongue only slightly if at all affected. (*c*) The leg type, in which the arm is paralysed to a lesser extent than the leg. This form is usually accompanied by hemianæsthesia, and probably also hemianopsia.

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These variations depend upon the position of the lesion in the centrum ovale or internal capsule. The general statement may be made that the more monoplegic the type of the palsy, the more likely is the lesion to be situated in or just below the cortex. Thus the facio-lingual type is due to a lesion corresponding in site to the lower part of the ascending frontal convolution, or the knee of the internal capsule; the arm type to the middle third of the Rolandic

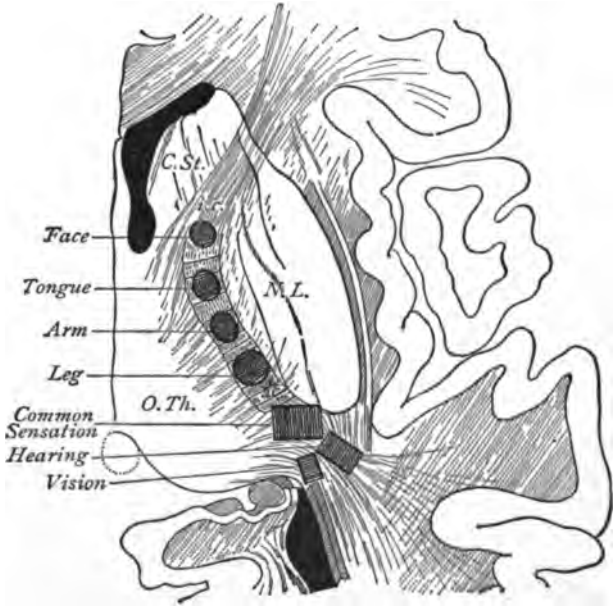

FIG. 7.—The internal capsule, showing the position of the afferent and efferent tracts. (Modified from von Monakow.) The circles represent the bundles of motor (pyramidal) fibres, the squares those of special sensation. *C. St.*, corpus striatum; *i. c.*, internal capsule; *N. L.*, nucleus lenticularis; *O. Th.*, optic thalamus.

area, or the anterior part of the posterior limb of the internal capsule; the leg type to the upper third of the Rolandic zone or to the middle of the posterior limb; while the associated hemianæsthesia and hemianopsia arise from destruction of the posterior and retro-lenticular parts of the internal capsule.

Muscular movements bilaterally represented, such as the trunk muscles, recover more rapidly, or are not paralysed at all, according to the extent of bilateral innervation; but this statement is not

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made without some reservation. According to this principle the movements of respiration and of the vocal cords are not as a rule implicated in cerebral lesions; the face may be more paralysed as regards voluntary than emotional movements, such as laughing and crying; the shoulder and hip movements are affected to a lesser extent than the more unilaterally represented movements of the wrists and ankles, while most paralysed of all are the fine and special movements of the hands and fingers.

Although the clinical course of a hemiplegia has been divided into two stages, there is no hard and fast line of demarcation, the late emerging gradually out of the early. The late stage is known as that of *chronic hemiplegia*. This is characterised by certain features to be now considered. Of these, the most obtrusive are rigidity and contracture, in which flexion predominates over extension, and pronation over supination. Hence the arm is flexed, the forearm pronated, and the fingers clenched; the thigh adducted, the knee rigid, and the toes pointed. The tendon and muscle jerks are exaggerated, an ankle clonus is usually obtained, and the plantar reflex is of the extensor type. In some adult hemiplegics athetosis, or mobile spasmodic movements of the paretic arm, are observed; but these are more commonly seen in the cerebral palsies of infancy and childhood. Closely allied in character, and probably of a similar nature, is the so-called post-hemiplegic chorea. Amongst other late phenomena are vaso-motor conditions, such as coldness, lividity, and sometimes œdema of the extremities of the paralysed limbs; lowering of the surface temperature on the paralysed side; and muscular atrophy arising from a unilateral peripheral neuritis and arthritic pain and swelling. Muscular wasting is more pronounced in the hand muscles, and especially in the abductor indicis. Alterations in the mental condition and in temperament are not uncommon; the memory is often markedly impaired, and the patient becomes unnaturally emotional, easily worried and hypochondriacal. It is, however, difficult to say how far the mental state is due to the lesion as such, or to the degenerative conditions, vascular or other, which co-exist.

Certain trophic disturbances are also observed, but they do not primarily belong to either the early or the late stages. In severe cases acute bedsores early occur; later on, in most cases, glossy skin, abnormal growth of the nails and of the hair on the paretic side, point to local inflammatory conditions of the peripheral nerves. Closely allied are arthritic troubles. Pain in the shoulder joint on movement often ensues soon after the stroke, and may exist for

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a long time. Less frequently affected in this way are the wrist, elbow, and knee joints.

Alternate paralysis, by which term is meant paralysis of the limbs on one side, and of a cranial nerve or nerves on the opposite side, will be described and its significance discussed in the section on Localisation, p. 131.

Double hemiplegia results theoretically from symmetrical and bilateral lesions in the cortex, subcortical white matter, or internal capsule, but complete paralysis from symmetrical, cortical, and subcortical lesions is undoubtedly rare; but the term has come to be modified with reference to the clinical facts of pseudo-bulbar paralysis, which is the chief example of bilateral hæmorrhagic lesions. In this the articulation is defective, the tongue only feebly protruded, the voice nasal, volitional movements of the lips and face deficient, and paresis of the extremities usually more marked on one side. This affection is usually associated with multiple hæmorrhages in the pons and medulla.

Prognosis.—The prognosis of the hemiplegic state depends largely upon the cause and its extent; the most favourable, both as regards life and recovery from paralysis, is that due to syphilitic endarteritis. In cases of this nature, in which the arterial occlusion is complete and permanent, softening results as certainly as in thrombosis from atheroma, with the result that the hemiplegia may persist.

In hemiplegia associated with arterio-capillary fibrosis and renal disease, several attacks, more or less complete, may occur before the final one which ends in death.

Hemiplegia from embolism in states of pyæmia is a serious complication of a commonly fatal disease. There does not appear to be any support to the view that right hemiplegia is more grave than left.

Treatment.—Much may be done, in the early weeks after the seizure, by careful and judicious movements to prevent the onset of the rigidities, contractures, and joint adhesions which characterise so many chronic hemiplegics. In order to be of much avail, however, the passive movements and exercises require to be continued over a long period of time.

In a favourable case passive movements of the paralysed limbs, associated with the application of galvanism and massage of the muscles, may be started within four weeks of the onset. These should, in the first instance, be carried out while the patient is lying in bed; but later on he may be trained in the use of simple move-

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ments of the limbs against slight resistance. Such indeed is the principle of the so-called Swedish movements, which are of much value in hemiplegia. Galvanism is of service in the prevention and reduction of rigidity in these cases, while faradism should be avoided throughout the whole period of treatment if there is proclivity towards rigidity and secondary contracture.

The pain of which the patient complains when the shoulder of the paralysed arm is moved, a more or less constant symptom of hemiplegia, is best treated by early movement at this joint. Should there be much adhesion of the joint it may be necessary to forcibly move it under an anæsthetic.

Medicinal remedies are of little or no value in the treatment of hemiplegia a few weeks after the onset, while all drugs likely to cause rigidity, such as strychnine, should be studiously avoided. It is scarcely necessary to do more than refer to the general dietetic and hygienic treatment of any underlying condition, such as chronic Bright's disease.

CEREBRAL PALSIES OF INFANCY AND CHILDHOOD

The clinical phenomena included under this term are produced by several pathological conditions, which occur shortly before, during, or at some time within the first few years after, birth. An exciting cause is not always to be found. In prenatal cases a maternal injury, or infectious disease during pregnancy, may be in part accountable; injury during parturition occasions a certain number, while after birth the commonest cause is to be found in infectious disorders, such as scarlatina, measles, and pertussis.

Morbid Anatomy.—The lesion is, in all cases, in the cerebrum, either on one or both sides. But as death is rare in the early stages, the late changes are those which have been chiefly studied. Therefore, although the pathological evidence of the early changes is by no means clear, it would seem as if the following conditions led directly to the clinical features to be immediately described:—(*a*) Simple mal-development; (*b*) Hæmorrhage (meningeal and cerebral); (*c*) Arterial thrombosis; (*d*) Diffuse encephalitis. From these, as pathological factors in the early stages, the two following conditions probably arise, and are observed in after years: (1) *Convolutionary sclerosis* (*microgyria*). This may be diffuse, or in patches. The convolutions are diminished in size, and microscopically there is found an absence or atrophy of the cortical cellular elements. (2) *Cavity formation* (*porencephaly*). This is a

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condition arising either from vascular thrombosis or from encephalitis. The wall of the cavity is formed of a dense fibrous membrane, which on the surface replaces more or less completely the convolutionary formation.

Clinically these palsies are met with as three distinct types: hemiplegia, diplegia, and paraplegia.

(*a*) *Hemiplegic type.* — This is characterised by paresis with rigidity, contracture, and mal-development of one side of the body, athetoid movements of the paretic limbs, and not infrequently tremors on voluntary effort. Sometimes there are motor and sensory aphasia and hemianopsia.

(*b*) *Diplegic type.*—In this are seen paresis, rigidity and contracture of the legs, paresis of the arms with athetoid or "choreiform" movements; weakness of the lower facial muscles may also be present. There is also exaggeration of the tendon jerks, and extensor type of plantar reflex.

(*c*) *Paraplegic type.*—Paresis and rigidity limited to the lower limbs, but often associated with some defect or irregularity in the movements of the arms.

These forms present certain additional features as a direct consequence of the palsies, rigidities, and contractures. Such are various forms of club-foot (talipes equinus and equino-varus). In the diplegic form the cross-legged progression may be seen; while in the hemiplegic variety athetoid movements of extraordinary violence have been observed in some cases.

To look a little more closely at these forms of disease as met with clinically, it should be pointed out that the diplegic and paraplegic forms are more usually characteristic of "birth palsy"; while the hemiplegic form occurs within the first two or three years of life.

In the BIRTH-PALSIES proper, the condition is detected within a few days after the birth of the child, and there may be a history of difficult or prolonged labour. As time goes on it is found that the child is unable to walk or stand; if he is able to progress at all, he does so with a rigid gait, the knee jerks are exaggerated and there is considerable adductor spasm. Movements of the arms are accompanied by athetoid spasm, depending upon the extent of implication of the upper limbs, and the finer movements of the hands are nearly always impaired. Tremors on volitional effort, such as are seen in disseminated sclerosis, may be present in some cases. Almost all present some degree of mental impairment, varying from a mere blunting of the intellectual faculties up to complete imbecility. Articulation also is not uncommonly affected.

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In the *hemiplegic form*, which occurs within the first two or three years after birth, the usual features of hemiplegia are detected. The onset is characterised by one or more convulsions, not necessarily limited to the side, which is afterwards found to be paralysed. Owing to the paralysis occurring early in life, the paretic side shows, when maturity is reached, marked atrophy, all the structures of the affected side being smaller and less well developed than those on the healthy side. Rigidity and contracture are common, as also is athetosis, as well as those vascular and trophic conditions which are seen in old-standing cases of adult hemiplegia. As in the birth palsies, there is often striking evidence of mental impairment; while epilepsy is a common symptom in these cases. This ensues in the majority, although its onset may not be until many years after the hemiplegia. The fits are of the usual epileptic character, but the convulsions are limited to, or more pronounced upon, the paretic side. In some instances 'le petit mal' is the type of seizure observed.

The **diagnosis** is not as a rule difficult. The history of difficult labour with onset of symptoms shortly after birth will, in the true "birth palsies," usually be sufficient to establish the nature of the condition. A careful inspection and palpation of the vertebral column should always be made to eliminate the possibility of spinal caries. A form of pseudo-paralysis associated with rickets may confuse the diagnosis in children at or about one year of age; while it is rare to find any difficulty in distinguishing this from infantile spinal paralysis.

Prognosis.—The presence of one or other type of "birth palsy" is in no way dangerous to life. Its influence is chiefly upon the growth and mental development of the child; the former showing itself in lateness in walking, the latter in backwardness, and general mental dulness and imbecility. These are more pronounced in the diplegic and hemiplegic forms. As already mentioned, in the latter variety the onset of epilepsy is to be regarded as certain in the majority of cases.

The **treatment** is conducted on general principles, until the child has reached an age when the question of education naturally arises. Unless the mental condition is average, these children should be educated separately from normal individuals. Physical training also is of much importance. Epileptic seizures are to be treated with the bromides; and deformities, if severe, upon orthopædie lines.

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ENCEPHALITIS

Encephalitis, or inflammation of the substance of the brain, is a condition the nature of which has only recently been determined. In times past the pathological appearances have been confused with the softening due to arterial occlusion or thrombosis. At the commencement of both inflammation and of softening the underlying changes are distinct; the effects, however, are similar.

Although considerable light has been lately thrown upon the pathological characteristics of inflammation of the brain, there is still some difficulty regarding a suitable classification. Pathologically it is divided into the *hæmorrhagic* and the *suppurative* forms; while clinically it is best described as *acute*—the varieties of which are focal and diffuse—and *chronic*, which is a patchy inflammation, giving rise to multiple or disseminated encephalitis.

It would seem as if all cases of encephalitis were due to a toxic or infective causation; but whether hæmorrhagic or suppurative depends upon the nature and source of the infection. The infective diseases with which the hæmorrhagic form is associated, whether focal or diffuse, are influenza, typhoid fever, diphtheria, syphilis, and probably the exanthemata. Certain toxic agents, the chief of which is alcohol, and more rarely lead, appear also as factors in the production of the hæmorrhagic form. Should, however, the source of infection be suppuration in the accessory cranial cavities—the nose, the orbit, and more especially the middle ear—the result is a suppurative encephalitis.

The whole drift of recent neuro-pathological investigation tends to the view that multiple sclerosis is a chronic inflammatory condition of infective origin, a conception of the disease which places it in line with such chronic degenerative conditions as tabes dorsalis, and general paralysis of the insane.

Acute (Focal) Hæmorrhagic Encephalitis

This may occur both in the gray and the white matter, but it is met most commonly in the gray matter of the mid and hind brain, involving the cranial nerve nuclei, and in the cerebral cortex. It is analogous to the acute anterior poliomyelitis of the spinal cord; both conditions arising from similar causes, and both having been known to occur in the same case at the same time.

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The pathological changes are thrombosis, hæmorrhagic extravasations, vascular distension, and leucocyte proliferation, with consequent destruction of the nervous elements. The lesion is characteristic of an acute inflammation, occurring as a sequence of the several infective diseases already mentioned, or having, as some believe, an infection peculiar to itself.

Clinically the onset is sudden, with general symptoms, such as vomiting, headache, convulsions, elevation of temperature, vertigo, and drowsiness. From these recovery may be partial, or the symptoms may deepen into coma, and death result. In some cases optic neuritis may be detected. In less severe forms the general symptoms are slight, the resulting paralytic effects being chiefly noted: for example, a sudden ophthalmoplegia (polioencephalitis superior of Wernicke) or its analogue, acute bulbar palsy (polioencephalitis inferior) indicating the nature and locality of the lesion.

Under this condition may also be classed some of those cases of sudden nuclear ophthalmoplegia occurring in the course of syphilis. In these the prognosis is essentially favourable under specific treatment. For this reason the underlying pathological state is undetermined.

It is believed, and some recorded cases would appear to bear out the view, that this condition may involve the gray matter of the cerebral cortex; but it is uncertain how far this may not be of the nature of the diffuse inflammation to be presently described. The **prognosis** depends mainly upon the severity of the initial phenomena, the most favourable as regards immediate recovery being those cases with slight or no initial symptoms. The residual effects are local palsies—ophthalmoplegia, bulbar palsy, or a complete or partial palsy of a limb.

A diffuse variety of this disease, in which hæmorrhagic foci are not limited to the gray matter of the third and fourth ventricles, or to the cerebral cortex, but are distributed over the greater part of the cerebrum, has been described. Associated with the hæmorrhagic infiltrations are obvious changes in the walls of the blood-vessels, consisting chiefly of small-cell infiltration. The clinical picture presented by this condition shows the patient, usually a child, becoming fretful and peevish; this is followed by a tottering gait and inability to walk, and frequently convulsions ending in coma. A common feature appears to be restlessness alternating with stupor. The reflexes are increased, and local palsies are rare. The result is not necessarily fatal, as has been seen in cases which have succumbed to a second attack.

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Acute Diffuse Encephalitis

Under this term is included a more extensive inflammatory condition of the brain than that just described. It is a rare condition, seen chiefly in infancy and childhood, the result of an acute infective disease, of severe traumatism. or, as some believe, of a special pathogenic organism, although there exists no conclusive evidence on this point. In adults syphilis is the principal cause, when it is usually combined with meningeal inflammation. This is the condition known as meningo-encephalitis, but as it presents specific characters, it will be separately described.

When met with in infancy and childhood, the immediate result is not necessarily fatal, but gives rise to one or other of the types of paralysis from cerebral causes seen in children—infantile hemiplegia and diplegia.

Pathologically large areas of the brain on one or both sides may be involved. Microscopically there are detected vascular dilatation, engorgement, ecchymosis, and leucocyte infiltration; these lead to extensive pulpiness, and eventually softening of the inflamed regions. Should recovery take place the affected portions in some are found materially atrophied, and in other cases cystic formation (porencephaly).

A diffuse meningo-encephalitis has been described by Dr. Mott, as the basis of the nervous symptoms in the rare condition known as Congo sickness (Vol. II. p. 34).

Suppurative Encephalitis—Abscess of the Brain

The ordinary form of this disease is a localised collection of pus due to organismal infection consequent upon suppuration in an adjacent accessory cavity, or from purulent conditions in remoter parts of the body. Any part of the brain may be affected, but that some localities are more prone than others may be seen from the following table from Mills, which shows the greater proclivity of the temporal lobe and cerebellum:—

Temporal lobe	40 cases
Cerebellum	31 „
Parietal lobe	7 „
Diffuse subdural	5 „
Pons	3 „
Frontal lobe	2 „
Other localities	10 „
	98 „

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The commonest source of infection is to be found in suppurative conditions of the mucous membrane of the accessory cavities, of which the middle ear is the chief. It is estimated that about 50 per cent of the cases of encephalic abscess are secondary to chronic middle-ear disease, acute otitis media being a relatively rare cause. Similar affections of the nose, frontal sinuses, antrum of Highmore, and orbit, though important, are infrequent sources. Cario-necrotic disease of the cranial bones and infected wounds of the scalp should be borne in mind in this connection. Injuries to the head, such as fracture and gunshot wounds, are also a frequent cause of intra-cranial abscess. The so-called metastatic abscesses of the brain are usually multiple, and are due to infection carried from distant parts of the body; such are those seen associated with—(*a*) a septic disease of the lungs, bronchi, and pleura; (*b*) septic peritonitis; (*c*) periostitis and osteomyelitis; and (*d*) pyæmia.

In the acute infective diseases—cerebrospinal meningitis, typhoid fever, erysipelas, and influenza—encephalic abscesses may also be found.

The bacteriological examination of pus in cases of brain abscess has shown the presence of streptococcus pyogenes and staphylococcus pyogenes aureus; in some instances Fraenkel's pneumococcus has been detected.

A brain abscess is usually situated in the white matter, and may or may not be separated from the surface by healthy nerve tissue. In the former case infection is carried by means of the perivascular lymphatics and venous sinuses. As a rule, however, there is a localised meningitis with adhesion between the brain and the dura mater, in which the extension is directly from the ear to the brain; while it is not uncommon for an abscess to be located between the dura and the bone (extra-cerebral abscess).

There is a close anatomical relation between the seat of the abscess and its source of infection. Abscesses arising from middle-ear disease are almost invariably situated either in the temporal lobe or in the cerebellum; the former being the locality if the superior wall of the middle ear is primarily affected, the latter if the posterior wall and mastoid cells are the seat of disease. Cerebral abscesses resulting from nasal and frontal sinus suppuration are almost always situated in the frontal lobe. In traumatic conditions the abscess is commonly located in immediate proximity to the cranial wound; while metastatic abscesses are multiple, but are more frequently seen in the basal ganglia than those from other causes.

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In some cases, more especially if the abscess be acute, the pus is present in the midst of broken-down nervous tissue; while it is only the chronic variety that is surrounded by a definite capsule.

Abscess of the brain is less common in early infancy and old age, the commonest period for its occurrence being during the second and third decades of life.

Symptoms.—A cerebral abscess may run an acute course lasting a few days or weeks, or it may remain latent for a long period, and eventually, through injury or other exciting cause, develop acute symptoms.

In all cases of chronic purulent otorrhœa presenting cerebral symptoms of sudden onset, a careful local examination of the ear, from which the discharge issues, should be made. Here will be found perforation of the tympanic membrane and a fœtid purulent discharge. In some cases granulations may be observed obstructing or even projecting through the perforation; in others the discharge is so slight as to be only recognised on introducing a cotton-wool mop into the meatus.

The general symptoms of an acute intracranial abscess may be roughly divided into the prodromal, those of full development, and those of the terminal period.

One of the earliest symptoms to attract the patient's attention is pain in the ear, commencing locally, but radiating over the side of the head. Vomiting is commoner in the early than in the late stages, unless the abscess be cerebellar, when it persists. Rigors are early apparent. The temperature may be slightly raised at this period, and the ear discharge usually ceases or very materially lessens. These symptoms last from one to three days, when they merge into those of a more pronounced type which characterise the stage of full development. Of these the most marked phenomena are a tendency to somnolence or lethargy, with excessive lassitude, loss of the power of attention, and inability to answer questions or carry on conversation. The temperature is normal or slightly subnormal; the pulse is slow and full, from 30 to 60 beats per minute; the respirations are slow and often of the Cheyne-Stokes type. Convulsions are rare unless the Rolandic area is directly or indirectly involved, but palsies are not uncommon, being due to pressure upon adjacent or even distant motor strands. Optic neuritis is often present, appearing, however, in the later stages. It is less severe than in cases of tumour; it is not invariably more pronounced on the side of the lesion, and it usually resolves upon opening the abscess.

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If surgical procedure is not carried out the terminal stage is reached. This is indicated by a deepening of the mental lethargy into stupor and eventually coma, in which the patient dies. To these may be added symptoms indicating co-existent complications, such as acute suppurative meningitis and sinus thrombosis, or the abscess may rupture into the ventricles.

As in cases of cerebral tumour, skull percussion has been used as a diagnostic agent; but as the diminished resonance which is present over an abscess may also be obtained over tumours or even hæmorrhagic effusions, its use is merely corroborative, the chief points in the diagnosis of cerebral abscess lying in the full recognition of suppuration in an accessory cranial cavity, and the presence of headache and increasing mental lethargy and stupor.

The two most common localities for intracranial abscess are the temporo-sphenoidal lobe and the cerebellum. Certain focal or localising symptoms are usually to be detected.

(*a*) *Temporal lobe.*—Macewen gives the following grouping of symptoms as characteristic of a large abscess in this situation :—(1) Paralysis of the third cranial nerve on the side of the ear disease as the result of pressure towards the base or from an associated meningitis. (2) Paralysis of the body on the opposite side, the weakness being greatest in the face, less in the arm, and least in the leg. Should the order of severity be reversed, the leg being most affected, there co-exists hemianæsthesia from pressure upon the internal capsule. In these conditions the facial palsy is of the cerebral type. Peripheral facial paralysis may be seen in some cases from interference with the nerve in the middle ear; in these cases the facial palsy is upon the same side as the abscess. (3) Word-deafness is pathognomonic of an interference with the functions of the first temporal gyrus upon the left side.

(*b*) *Cerebellum.*—The general symptoms of cerebellar implication are seen in retraction of the head and rigidity of the neck muscles, giddiness, and a staggering gait, so that the patient falls to one or other side. Paresis of the limbs on the side of the lesion has been observed in some cases. Some patients assume in bed a characteristic attitude, lying on the side with the limbs flexed, and the side of the face on the same side as the abscess uppermost. The knee-jerk on the side of the abscess may or may not be exaggerated.

(*c*) Abscesses may occur in other localities, *e.g.* the *frontal lobe*, as a result chiefly of rhinitic suppuration (ethmoid, sphenoid and frontal sinuses). If the abscess is of any size, the symptoms are

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aphasia, if situated on the left side, and paralysis of the limbs on the opposite side from pressure upon the third frontal gyrus and the Rolandic area respectively. Much more rarely are the basal ganglia, crura cerebri, and pons the seat of suppuration.

In some cases where the symptoms pointed to a localised collection of pus the autopsy revealed a diffuse subdural purulent condition. Should this be basal the cranial nerves are implicated, but if upon the convexity it may be difficult to form any precise opinion as to the locality, as focal symptoms are rarely present.

Latent abscess.—Such a condition may present no symptoms whatsoever; on the other hand some irritability, a tendency towards inertia, with headache and an occasional convulsion may serve to attract attention. These symptoms may entirely pass away, and months or years later an injury to the head or a relapse of a forgotten ear trouble may be productive of acute cerebral symptoms to which the patient succumbs.

Diagnosis.—As will be readily seen, the diagnosis of intracranial abscess is often difficult, but in no encephalic condition are the results of a correct diagnosis and appropriate surgical treatment more striking and satisfactory. In forming a diagnosis too great importance cannot be placed upon a full investigation into the antecedent history, especially with reference to aural and nasal suppuration and trauma.

POINTS IN THE DIFFERENTIAL DIAGNOSIS BETWEEN INTRACRANIAL ABSCESS, SINUS THROMBOSIS, AND MASTOID SUPPURATION.

ABSCESS.	SINUS THROMBOSIS.	MASTOID DISEASE.
Mental symptoms common and characterised by hebetude, lethargy, and somnolence	(If uncomplicated) absence of mental and focal symptoms	Headache severe, intermitting, and radiating from the mastoid.
Focal symptoms indicate lesion of temporal lobe, cerebellum, or frontal lobe	Pain, tenderness, and hardness in retromaxillary region and along jugular vein	Pain and tenderness, redness and œdema over the mastoid.
Temperature normal or subnormal	Temperature of pyæmic character with rigors and vomiting	Slight pyrexia or elevated to 104° or 105° F.
Pulse slow	Pulse rapid and small	...
Optic neuritis absent or late	Optic neuritis may be present	(If uncomplicated) optic neuritis absent.
		State of the ear—rapid flow of pus after cleansing, swelling of "the dip."

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The differential diagnosis has to be made from intracranial tumour, meningitis, lateral sinus thrombosis, and mastoid suppuration. The points in the differential diagnosis between lateral sinus thrombosis, mastoid suppuration, and abscess are given in the foregoing Table; but for the distinguishing features of tumour and meningitis the reader is referred to the Table upon p. 112.

It may be a matter of extreme difficulty to decide, in a case of middle-ear suppuration, whether the onset of headache, often severe, and radiating over the head, and sometimes accompanied by vomiting and attacks of rigidity, is due to the incidence of serious intracranial disease, or is merely associated with the local disease of the ear or mastoid suppuration. The points to be specially borne in mind are the absence of the characteristic mental lethargy and somnolence and the existence of pyrexia in the local affections.

Prognosis.—Though necessarily grave, the prognosis of cerebral abscess is more favourable than in many other intracranial conditions. If left untreated surgically, death usually ensues, though some abscesses are known to become latent, more especially if chronic. The presence of a latent cerebral abscess is a source of great danger, as it may suddenly assume an acute character from slight causes or give rise to suppurative meningitis. Even if surgically treated, a patient with cerebral abscess may succumb to pyæmia, sinus thrombosis, or pulmonary disease. The successful results of recent operative procedures in uncomplicated cases have largely ameliorated the prognosis.

Treatment.—From what has already been said of the causation of intracranial abscess, the systematic and judicious handling of chronic suppuration of the accessory cranial cavities and sinuses is of primary importance. Although the recognition of this principle, so far as the ear is concerned, is now universal, it is less attended to in chronic nasal conditions and frontal sinus suppuration, though the morbid processes are similar in both diseases. Hence the importance of prophylaxis in suppurative encephalitis cannot be over-estimated.

In regard to abscesses resulting from fracture of the skull, or scalp wounds involving the periosteum, the application of the methods of antiseptic surgery are naturally all-important.

Should the diagnosis of intracranial suppuration be definitely made, the treatment at once becomes an urgent surgical matter, the principles guiding the procedure being—immediate opening of the skull over that portion of the brain which the focal symptoms indicate as the seat of the abscess, and the removal of sufficient

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bone to ensure adequate evacuation and drainage. The question of opening the skull when focal symptoms fail, though general symptoms indicative of intracranial suppuration are present, may be answered in the affirmative, the locality chosen for the trephine opening being over that part of the brain which stands in relation to the local source of infection.

INTRACRANIAL SYPHILIS

Syphilis may affect the cranial contents in two ways, first by the production of vascular lesions, which take the form of obliterative endarteritis and gummatous formations, and secondly by favouring the development of degenerative diseases. The vascular lesions are early manifestations of the action of the syphilitic poison upon the cerebral circulatory system, while the degenerative diseases are essentially late phenomena. This division of the effects of the syphilitic poison upon the cranial contents is more in harmony with modern doctrines than that which regards certain phenomena as belonging to the "secondary" and others to the "tertiary" stage. For cerebral symptoms may occur within a comparatively short time of syphilitic infection, a recent statistical account showing that 44 per cent of the cases of cerebral syphilis occurred within the first three years, and 59.5 per cent within the first five years following infection. Cases are indeed on record in which syphilitic cerebral lesions have ensued while traces of the indurated chancre still existed; and one case is described in which hemiplegia occurred forty-six days after the first appearance of the disease. It is therefore clear that, so far from being an indication of the deferred action of the syphilitic virus, symptoms pointing to intracranial disease are in at least half the cases evidence of an early affection of the blood-vessels and membranes of the brain.

Some other facts of etiological interest may be briefly mentioned in connection with the incidence of cerebral syphilis. The statement is sometimes made that those who suffer from nervous symptoms in the later stages of syphilis have passed through a mild form of the disease in the earlier. This is not a constant feature, for nervous symptoms may arise even after prolonged antisyphilitic medication, while the onset of cerebral symptoms is not unknown even during the administration of the remedies.

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There is no clear evidence that a hereditary disposition to mental or nervous disease favours the occurrence of intracranial disease in those who have acquired syphilis. A neurotic tendency seems rather to influence the type and course of the disease than to determine its occurrence. In the majority of cases of intracranial syphilis, the immediate causes cannot be ascertained, the meningeal and vascular affections being apparently spontaneous.

In the present chapter the early syphilitic affections alone will be considered, the description of general paralysis of the insane, a degenerative or parasyphilitic disease, being reserved for a later page.

Morbid anatomy.—The syphilitic affections of the intracranial contents are (1) lesions of the blood-vessels, (2) lesions of the brain and its membranes. The chief vascular lesion is the *obliterating endarteritis*, which is said to be characteristic of this disease. This is not absolutely correct, for it is found associated with most chronic inflammatory conditions, such as tubercle, and it is also seen in the neighbourhood of gliomatous tumours. It chiefly implicates the cerebral arterioles, causing a uniform diminution, and eventually obliteration of their lumen with thrombosis. The vessels chiefly affected are certain of the cerebral arteries—the basilar and vertebrals, the middle cerebral and its branches, and more rarely the vessels entering the basal ganglia. Endarteritis obliterans rarely occurs alone, being usually associated with a small-celled infiltration of the adventitia, giving rise to the condition of periarteritis. In this arise the gummatous nodules so commonly associated with syphilitic cerebral disease. Complete obliteration of the lumen at the onset of cerebral syphilitic disease is rare, as will be shown when the clinical phenomena of this disorder are considered, when transitory and fleeting paralysis point to partial obstruction and local ischæmia. This partial obliteration, however, may eventually end in complete closure of the vessel and consequent cerebral softening. A form of *amyloid or waxy degeneration* of the middle coat of the cerebral arteries occurring in syphilitic cases has been described, but it is a rare condition. It has been found associated with patches of degenerated nervous elements, giving the amyloid reaction, disseminated throughout the central nervous axis. Atheroma may be of syphilitic nature, but is more commonly a degenerative disease, predisposed to by gout, and associated with chronic renal cirrhosis.

The other early syphilitic lesion is the *gumma* or *gummatous meningo-encephalitis*. The commonest situations for this are the convexity of the cerebrum, the base of the brain, the walls of the

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arteries, the interior of the brain, and on the cranial nerves. The gumma may be single or multiple, and varies in size up to a pigeon's egg. Growing in the subarachnoid space it spreads along and into the brain, causing adhesion between the membranes and the convolutions. The gumma is composed of small round and spindle-shaped cells aggregated into masses which tend to become caseous.

A variety of the gumma is seen in the *diffuse gummatous meningo-encephalitis*, also a characteristically syphilitic formation. In the early stages it is of gelatinous consistence, but later on assumes a fibro-caseous appearance. A chronic form of meningeal thickening, chiefly over the base of the brain, is also met with as a syphilitic lesion, and is a common cause of paralysis of the cranial nerves.

Symptoms.—Although the term "cerebral syphilis" is used to denote a series of symptoms arising from the morbid changes produced by the action of the syphilitic virus upon the blood-vessels of the brain, it may be stated as a general principle that the phenomena so produced are in no way peculiar to this disease.

Thus the hemiplegia arising from syphilitic endarteritis, the localised convulsions of gummatous origin, or the paralyses of the cranial nerves resulting from a syphilitic basal meningitis do not differ in character from like symptoms arising from causes of a non-syphilitic nature. But although it cannot be stated that cerebral syphilis is characterised by any symptom which may be regarded as pathognomonic, there exist associations of symptoms suggestive of this condition, of which the following may be mentioned:—(1) An association or succession of symptoms indicative of double or multiple lesions; (2) symptoms having a tendency to remission and relapse; (3) symptoms having an onset which is sudden rather than acute, and a course which is subacute or subchronic rather than chronic; (4) symptoms which, as a rule, resolve rapidly under appropriate treatment; but it should be pointed out that some cases of cerebral syphilis are uncertain and variable under antisyphilitic remedies.

Various prodromal symptoms usually herald for a longer or shorter period the onset of graver cerebral phenomena. Of these the most common are headache, characterised by a tendency to increase towards evening and the early hours of the morning; insomnia, vertigo, and mental apathy. Other initiatory phenomena, sometimes of a local, at other times of a psychical character, have been observed. Of these may be mentioned, epileptiform con-

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vulsions, verbal amnesia, attacks of confusion of mind without palsy or aphasia, epileptic fits, melancholia, and acute mania.

Clinical forms.—The symptoms arising from cerebral syphilitic lesions may be conveniently studied under the following heads: symptoms due to vascular occlusion — hemiplegia and aphasia; symptoms due to gummata of the membranes and cortex—Jacksonian epilepsy and monoplegia; gummatous meningitis of the membranes of the base—paralysis of the cranial nerves; diffuse arterial and meningeal lesions—syphilitic dementia.

1. *Symptoms arising from vascular occlusion.*—Of these the two most important are hemiplegia and aphasia. Syphilitic hemiplegia, though differing in no respects from that due to other causes, presents certain features which suggest its syphilitic nature; thus its onset is rarely attended by unconsciousness, it seldom occurs without warning, it is usually of a temporary character, and after recovery it may be followed by a second hemiplegia upon the same or opposite side of the body. Of a like nature, and arising from a similar cause, are the following symptoms, which may either precede an attack of hemiplegia, or may for a time form the only evidence of endarteritis: transient and incomplete hemiplegia and aphasia, unilateral numbness and paræsthesia, verbal amnesia or inability to find the right word, temporary lapses of consciousness and confusion of mind.

Hemiplegic seizures of syphilitic causation are not always transient. If the vascular occlusion is complete and permanent, cerebral softening may result and the hemiplegia differs in no respect from that seen under other conditions and already described in detail. These are the cases in which anti-syphilitic treatment is of little or no avail.

Cerebral hæmorrhage arising directly from syphilitic vascular disease is rare, unless through the rupture of an aneurysm, in which case the basilar is the artery most commonly affected.

2. *Symptoms due to gummata of the cortex and cortical membranes.*—Due to this cause are the partial, localised, or Jacksonian convulsions, and monoplegia or hemiplegia of cortical nature. Epileptiform attacks of syphilitic causation are similar to those due to any other cerebral tumour in a like locality. But loss of consciousness is rare, or, if present, seldom complete. They are usually accompanied by mental apathy and the loss of memory and attention common in cerebral syphilis. The paralyses from gummatous new growth differ from those caused by vascular occlusion in their more gradual incidence and longer duration. Gummatous disease

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is associated with optic neuritis, which is characterised by its early appearance, rapid onset, and great intensity.

Various forms of aphasia may be due to gummatous cortical disease; thus motor aphasia may be associated with unilateral convulsion and right-sided hemiplegia, or word-blindness and word-deafness may arise from vascular and gummatous lesions of the angular and superior temporal convolutions respectively. Whether true epilepsy is ever directly traceable to the direct influence of the syphilitic poison is a moot point; but that seizures, differing in no respect from those of idiopathic epilepsy, may follow vascular occlusion, or the cicatricial remnants of a healed gummatous new growth, is well known.

3. *Symptoms arising from gummatous meningitis over the base of the brain and basal meninges—paralysis of the cranial nerves.*—All the cranial nerves, motor as well as sensory, may be affected from this cause. The commonest lesions are those of the oculo-motor nerves, the third and the sixth. Not only may oculo-motor paralysis of peripheral origin be due to this cause, but also those more complete forms of ophthalmoplegia — paralysis of all the external ocular muscles—may be ascribed to vascular occlusion in the arteries supplying the gray matter around the Sylvian aqueduct. The olfactory and optic nerves are rarely involved; but a bi-temporal hemianopsia may be observed from gummatous lesion of the interpeduncular space. Paralysis of the trigeminal, facial, and auditory nerves may all, or individually, be involved in meningeal thickening; while a paralysis of the bulbar nerves—vagoglossopharyngeus and hypoglossus—although not frequent, may likewise arise from the same cause.

Cranial nerve paralyses may exist alone, or in association with graver cerebral symptoms, such as vertigo, hemiplegia, localised convulsions, and optic neuritis.

4. *Symptoms arising from diffuse arterial and meningeal lesions*—Syphilitic dementia. — The mental symptoms, often obtrusive in cases of cerebral syphilis, are not necessarily due to any particular form of syphilitic lesion. Their co-existence with various local palsies, or with hemiplegia or aphasia, or with epileptiform seizures, suggests the presence of diffuse vascular and meningeal affections.

Although it may be difficult to distinguish, at all events in the early stages, the mental and motor phenomena of cerebral syphilis from true general paralysis of the insane, it may be stated that the former show a more marked tendency to resolve under general antisyphilitic treatment.

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Of the mental phenomena of cerebral syphilis the following may be mentioned, although they are not necessarily characteristic of this disease, but their association with more purely motor symptoms is highly suggestive. Such are, loss of memory and a failure of the power of attention, attacks of confusion of thought, verbal amnesia and temporary lapses of consciousness, with or without subsequent temporary aphasia, a curious tendency to somnolence, verging at times into actual stupor, restlessness, delusions and melancholia, and acute mania.

Prognosis.— If there be one cerebral affection more than another in which each case requires to be studied and judged upon its merits, it is intracranial syphilis, for so much depends upon the structural alterations which have occurred. In the early stages of the disease suitable remedies may be of immense value; but if fibroid transformation or caseation has taken place, or if the tissue has become softened from vascular occlusion, little benefit is likely to accrue. Gummata in the early stages or gummatous meningo-encephalitis are especially favourable for treatment; cranial nerve palsies from basal meningeal lesions are also of satisfactory prognostic nature, while thrombosis which has led to softening is unfavourable so far as actual or immediate recovery is concerned.

From various statistical data it has been shown that thirty to thirty-five per cent of cases of cerebral syphilis recover, that about twelve per cent die, while the remainder are more or less permanently maimed. There does not appear to be any material difference in the percentage of cured and uncured cases, whether considered from the age at which the symptoms manifest themselves, or whether they occur within a short period of infection or after an interval of ten years.

In estimating the prognosis of cerebral syphilis, the tendency to relapse and remission should always be borne in mind. As already indicated, this is a characteristic feature of the disease, and as such plays an important part in prognosis.

Treatment.—There are no rules for the treatment of syphilis generally which are not applicable to the treatment of syphilitic affections of the central nervous system, attention being here merely directed to a few points concerning the methods of administration of the antisyphilitic remedies.

Of the first importance is placed *mercury* or its preparations. It may be given in various ways: by fumigation, by inunction, by injection or by baths, and internally. The continuous administration of mercury *by the mouth*, either in the form of calomel or corrosive sublimate, may lead to troublesome forms of intestinal

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derangement; and it is a matter of importance that the kidneys should be in a sound state for this treatment.

Inunction is largely practised at the spas to which syphilitic patients resort. This method has distinct disadvantages in that it often occasions troublesome forms of stomatitis and gingivitis. Its chief objection lies in the fact that although a definite amount of mercurial ointment may be rubbed into the axillæ or the groins, there is no criterion that it is all absorbed. Fournier recommends that from 5 to 10 grammes (approximately 1 to 2½ drachms) may be rubbed in *per diem*; while a judicious rule is to rub in that amount of mercurial ointment, a small increase to which will produce salivation. It is difficult to fix a period for the carrying out of this treatment; but 50 to 60 inunctions are usually sufficient. *Intramuscular injections* of various of the soluble mercurial salts, perchloride, peptonate, salicylate, and sozoiodol are strongly advocated by some writers. The chief advantage of this method is that a known quantity of the drug is given, all of which is absorbed; hence smaller doses are sufficient, and a less frequent application is necessary (one injection per week being counted sufficient by those who use the method). The old method of *calomel baths* is not resorted to at the present time, owing to troublesomeness of its application, but it is a useful method.

Next in importance come the *iodides of potassium, sodium, and ammonium.* The dosage of these drugs varies considerably in different countries. Thus in America enormous doses are administered—120 to 150 grains of iodide of potassium thrice daily being no uncommon amount. In France doses of considerably less magnitude are given, but on the whole larger than what is customary in this country, *e.g.* 2 to 10 or 12 grammes *per diem* (30 to 180 grains). In England the dosage also varies within considerable limits. Some physicians are content with 5 to 7 grains thrice daily; others consider that 15 to 20 or 30 grains of potassium iodide given thrice daily or, better still, every six hours will produce as marked benefit as the larger doses already mentioned. There is one important point to be noted, viz. that *iodism* is much more likely to occur from the use of small than from large doses.

Perhaps the most beneficial method of internal administration is the combined use of mercury and the iodides in the form of liquor hydrargyri perchloridi (B.P.) with the iodides of potassium, sodium or ammonium. The combined method may also be carried out by giving iodide internally in conjunction with mercurial inunction.

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A word may be said as to the use of *natural baths* in the treatment of cerebral syphilis. At those spas to which syphilitic subjects most resort, the waters are of a "sulphurous" nature and hot, but in addition to the natural baths treatment by inunction and by iodide is carried out. It appears that the sulphur baths hasten the elimination of mercury from the system, thus enabling larger doses of mercury to be given.

GENERAL PARALYSIS OF THE INSANE

General paralysis, or paretic dementia, is a progressive degenerative disease of the central nervous system characterised by motor as well as by mental symptoms, and in the majority of cases is the result of a previous syphilitic infection. The evidence bearing upon the syphilitic causation of general paralysis is accumulating. Much statistical information is at hand showing the percentage of general paralytics in whom a clear history of acquired syphilis has been obtained. The figures given by several authorities, both in this country and abroad, vary from 75 to 86 per cent, a ratio which has a close correspondence to that of locomotor ataxy, a disease which is believed to stand in much the same relation to syphilis as general paralysis. It would, therefore, appear as if the influence of acquired syphilis was the great predisposing cause of this disease. Of other factors in its causation, which probably act only as excitants, the following may be stated :—

i. Chemical poisons.—Alcohol in excess, although productive of mental as well as motor nervous disease and degeneration of the neurone, will not alone give rise to general paralysis, nor will other chemical poisons, such as lead, although the clinical appearances both in alcoholic and lead encephalopathy are difficult to distinguish from true general paralysis.

ii. Mental worry and anxiety and sexual excesses.

iii. Trauma would seem in some cases to be instrumental in exciting the disease in those already disposed to it by syphilis ; under this may also be included sunstroke, which is not infrequently a determinant.

General paralysis is much more common in men than in women. Of the latter, it is more frequently found in the lower social ranks than in the upper classes. It is more common in urban than in

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rural districts. It is more frequent between the ages of thirty-five and fifty than either before or after these ages, although a juvenile form of general paralysis, the result of inherited syphilis, is observed during puberty and in adolescent life. It is rarely associated with any family disposition to nervous or mental disease.

Pathology. — General paralysis is a typical instance of a degeneration affecting certain neuronic systems as the result of the action of a toxic agent, that of syphilis; but whether the degeneration is primary, as maintained by Dr. Mott, or is the result of a co-existing vascular degeneration, as held by some observers, is not yet clear.

To the naked eye, the brain of a general paralytic is shrunken and atrophied, more especially over the frontal and Rolandic regions, the convolutions in these localities being small and the pia-arachnoid membrane thickened and frequently adherent to the subjacent gray matter, so that it is not possible to strip it off freely, as in the normal condition. Occupying the subdural space, there may be found blood clots in various stages of absorption and organisation, or cystic formations giving rise to the condition known as pachymeningitis hæmorrhagica (p. 52). The left hemisphere may weigh considerably less than the right; the ventricles are distended and contain an excess of cerebrospinal fluid, and the ependyma ventriculorum usually presents a granular appearance. Atheroma of the larger blood-vessels may also be seen.

A microscopic section of the cortex cerebri in an early and typical case of the disease shows degenerative changes in the walls of the cortical arterioles, such as thickening of the intima, hyaline and fatty morphosis, and proliferation of nuclei in the perivascular spaces. The glia cells are increased in number and are prominent features; while numerous, deeply-stained nuclei are observed in the section. The cortical pyramidal cells show the changes already described under degeneration of the neurone (p. 36). There is degeneration of the fine medullated fibres of the convolutions, the tangential and association fibres of some observers. Associated with these purely cortical changes may also be seen thickening of and inflammatory alterations in the superjacent pia-arachnoid membrane.

The evidences of degeneration, both vascular and nervous, are not limited to the cerebral cortex. Similar alterations are found in the subcortical tissues, the basal ganglia, the pons, the medulla oblongata, the spinal cord, and the peripheral nerves.

In the spinal cord may be noted in some forms of the disease

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a descending degeneration in the crossed pyramidal tracts (degeneration of the upper efferent neurone), while in other cases the posterior columns are affected by a degeneration similar to that seen in tabes dorsalis (degeneration of the lowest afferent neuronic system). (*Vide* Plate III.)

From the morbid appearances found in this disease, two theories have been advanced as to its pathogeny. First, that it is a meningo-encephalitis, which leads to a secondary degeneration of the neurone, a view held by many of the older observers. Secondly, that it is a primary neuronic degeneration, to which the vascular and other phenomena already described are secondary.

Symptoms.—Various types of the disease are met with clinically, a rough classification being made into those as seen originally on the one hand in asylums for the insane and on the other in hospital practice. The former class is characterised by the obtrusiveness of the mental phenomena; the latter by the predominance of the motor symptoms, but in this class there also exists considerable mental failure, such as loss of memory and facility of the mind, with (less commonly) the existence of grandiose ideas.

The following five types may be taken as illustrative of the commencement of the disease:—

(1) *The maniacal type.*—This takes the form of exaltation with grandiose ideas, the patient in many cases acting up to his delusions, as, for instance, spending large sums of money. He is restless, agitated, laughing, or singing. About 50 per cent of the cases begin in this way.

(2) *The melancholic type.*—This is rarer than the preceding, but at the same time presents a very definite clinical picture. It usually commences with neurasthenic symptoms which precede for a longer or shorter period the onset of obvious melancholia. This may be characterised by all the phenomena common in melancholy, such as delusions that he is being poisoned, refusal of food, etc.

(3) *The motor type.*—Of this there are usually seen two varieties, the spastic and the ataxic, the former characterised by tremors of the tongue, lips, and face, slurring of the articulation, exaggeration of the knee-jerks, loss of the pupillary light reflex and a spastic gait; the latter by ataxia, loss of the knee-jerks, and sometimes perforating ulcer on the sole of the foot. An examination into the mental condition in both forms will usually show a considerable amount of mental facility bordering almost upon dementia with impairment of memory, while the facial expression may be described as dull and fatuous.

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(4) *The convulsive type.*—Here the disease commences with a series of one or more general epileptic seizures. Upon recovery, some mental or motor impairment may be detected, but in many instances the patient returns to his usual condition of health, in which he remains until he is prostrated by another series of convulsions.

(5) *Juvenile general paralysis.*—A condition occurring during puberty and in early adolescent life, having chiefly the symptoms presented by the motor type as seen in adults.

In whichever of the above described ways the disease originally commences, certain definite symptoms which denote the true nature of the malady may later on be detected in all cases:—

(*a*) Dementia characterised by impairment of memory, weakness of will power, the retention of obvious, but modified, grandiose ideas, facility of mind, and a fatuity and flabbiness of facial expression.

(*b*) Motor symptoms, taking the form of facial tremor upon voluntary and emotional movements, tremulous handwriting, unsteadiness of gait, and a characteristic slurring of the articulation. In some cases the knee-jerks are exaggerated; in others they are abolished.

(*c*) Epileptiform convulsions, the so-called "congestive attacks" of some authors. These may be localised or general. Their frequency varies in different cases, but if frequent they lead rapidly to increased dementia and a fatal issue. During the attack, the convulsions are of a clonic type. The tongue may be bitten and the excreta passed involuntarily. The temperature is raised sometimes to hyperpyrexia.

(*d*) As intercurrent phenomena may be mentioned a readiness towards fracture of the bones and aural hæmatoma.

From this it will be apparent that the disease is a progressive mental enfeeblement with motor weakness, the whole ending in dementia and paralysis. Three stages of the disease are described, but this subdivision seems scarcely necessary owing to the enormous variation of type met with in practice, but the difference between the symptoms of the early stage, with its grandiose ideas and its extravagance, and the final stage, in which the patient lies demented and paralysed, passing his excreta involuntarily, at times the subject of severe and prolonged convulsive seizures, are two aspects of a clinical picture not easily forgotten. Remissions are not infrequent, the temporary mental disturbance often clearing away, but leaving some general mental enfeeblement. The transitory disappearance

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of the mental symptoms may throw into relief the characteristic motor phenomena which establish the diagnosis of the disease.

Prognosis.—The duration of the disease is, roughly, from three to five years, but more commonly the former. No disease is more uncertain in its course and duration than general paralysis. A convulsive seizure may happen at any time and carry the patient off, or he may rapidly pass into dementia and paralysis, in which death occurs.

Diagnosis.—For the specific diagnosis of this disease, whether the mental phenomena are those of neurasthenia, melancholia, or acute mania, the presence of certain motor symptoms is essential, such as the reflex pupillary immobility, exaggeration or abolition of the knee-jerks, tremors of the face and tongue, blurring of the articulation, and convulsive seizures. Should such mental and motor phenomena be combined with a history of acquired syphilitic infection, general paralysis is the disease with which the physician has to deal. The disease may have to be distinguished from—

Cerebral syphilis.—This condition, as already described (p. 96), usually presents definite local paralyses, such as cranial nerve palsies, or paralysis of a limb or limbs, which phenomena are against the existence of general paralysis.

Chronic alcoholism.—One of the most difficult problems in clinical neurology is the differentiation of this condition from general paralysis, as all the phenomena of the latter, both mental and motor, may be simulated in alcoholic poisoning. The chief point, however, lies in the absence of pupillary immobility and in the normal retention of the knee-jerk.

Lead encephalopathy.—In this there are usually other signs of lead poisoning, such as a blue line upon the gums, weakness of the extensor muscles of the fore-arms, and in many cases double optic neuritis.

Treatment.—In cases presenting obvious mental phenomena, removal to an asylum is the only satisfactory procedure. Should it be possible to treat the patient at home, on account of the absence of definite delusions, rest is absolutely essential. Travelling abroad or a sea voyage are methods not likely to lead to any permanent benefit. It is preferable that the patient should be sent to a quiet place in the country, where there is avoidance of excitement and the possibility of alcoholic or other excesses.

As regards medicinal treatment, drugs are of practically little avail. Should there be a clear history of comparatively recent syphilitic infection, and should the patient have not been treated

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systematically at the time, a course of mercury and iodide of potassium is often of temporary benefit. The symptoms must be treated as they arise. Sleeplessness must be counteracted by the usual remedies, paraldehyde, sulphonal, or trional. Convulsions require the administration of the bromides, while in the late stages careful nursing and general attention are necessary.

TUMOURS OF THE BRAIN

General characters.—Encephalic tumours are of three kinds:—(1) The infective granulomata—tubercle, gumma, and actinomyces; (2) true neoplasms—sarcomata, gliomata, carcinomata, and the several benign forms; (3) the parasitic cysts.

Tumours are found within the cranial cavity springing from the bone, the membranes, the neuroglia, and the adventitia of the blood-vessels. As contrasted with other organic diseases of the central nervous system, encephalic tumours are relatively rare; on the other hand, the brain is one of the most frequent sites of tumour formation in the body. Some new growths are commoner than others at certain periods of life, so also some forms of tumour affect some regions of the brain more constantly than others. Men are more frequently the subjects of cerebral tumour than women. The greatest number occur in childhood and youth, and in old age they are relatively rare.

Of the INFECTIVE GRANULOMATA *tubercle* is the one which is most commonly seen on the post-mortem table. Tuberculous tumours vary in size from a pea to a small orange; they are usually multiple; they are often encapsulated and may be readily enucleated; as a rule they undergo caseous degeneration in the centre, and it is most probable that, like the gummata, they spring from the outer covering of the blood-vessels. Their most frequent locality is the cerebellum, but they are also common in the cerebral hemispheres. The *gummata* are the most common form of new growth, though, owing to their greater curability, they are less frequently observed post-mortem than tubercle. Though not presenting a definite capsule, they do not infiltrate the cerebral substance; they are irregular in shape and variable in size, and the cut surface presents a gelatinous appearance. As the result of treatment a fibrous cicatrix is often all that is seen of a previous gummatous mass. They would appear to

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spring in all cases from the adventitia of a blood-vessel. They are most commonly seen in the cerebrum. They do not occur without other indications of syphilitic infection, such as endarteritis.

The following table, based upon Starr's statistics from post-mortem records, shows (*a*) the relative frequency of the several forms of intracranial new growths (*b*) at different ages and (*c*) in different localities, as indicated by their numerical order:—

No.	Nature of Growth.	In Children up to Puberty.	In Adults.[1]
1.	Tubercle.	1. Cerebellum. 2. Pons. 3. Corpora quadrigemina. 4. Basal ganglia.	1. Pons. 2. Cortex cerebri. 3. Cerebellum.
2.	Sarcomata.	1. Cerebellum.	1. Cortex cerebri. 2. Cerebellum. 3. Basal ganglia.
3.	Gliomata.	1. Cerebellum. 2. Pons. 3. Cortex cerebri.	1. Cortex cerebri. 2. Centrum ovale. 3. Basal ganglia. 4. Cerebellum.
4.	Cysts.	1. Centrum ovale. 2. Cerebellum.	Rare.
5.	Carcinomata.	1. Cerebellum.	1. Cortex cerebri.
6.	Gummata.[2]	Rare.	1. Cortex cerebri.
7.	Other tumours.	1. Cerebellum. 2. Centrum ovale.	1. Cortex cerebri. 2. Cerebellum.

TRUE NEOPLASMS.—The *sarcomata* form an important group of encephalic tumours. They are usually single. Some are hard, fibrous, and encapsulated; others are soft and infiltrating, and in some cases cystic. The former are characterised by round or spindle cells and a variable amount of intercellular staining material; the latter by a minimal amount of intercellular matter. These are probably of a gliomatous nature, and in them may be found large spider-shaped cells. It is probable that the two kinds of growth

[1] Tumours after sixty years of age are rare.

[2] According to post-mortem records; clinically the gummata are common, but, being susceptible to treatment, are less frequently seen after death.

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may be found together forming a glio-sarcoma. Owing to the variations in the intercellular matter, and the degenerative changes which may occur in the new growth, many varieties of sarcoma are recognised; such are the fibro-sarcoma, angio-sarcoma, myxo-sarcoma, melanotic sarcoma, alveolar sarcoma, and cystic sarcoma. In the more vascular and cellular forms, hæmorrhage into the substance of the tumour is not uncommon.

The sarcomata may be found in any part of the brain, but they commonly spring from the membranes and compress the cerebral tissue; the gliomata affect the cerebellum and cerebrum to about an equal extent. Owing to the infiltrating character of these tumours, an appearance of hypertrophy of the affected part is produced.

Carcinoma is a relatively rare form of intracranial tumour. It may be primary, but is more usually secondary to cancerous formation elsewhere. It is, as a rule, single, but may be multiple, and even symmetrical. The cerebral hemisphere is its commonest site.

The *benign tumours* are rare inside the skull. New growths having a fibrous texture and appearance are, as a rule, sarcomatous in character. Bony tumours are sometimes seen growing from the base of the skull, and involving the cranial nerves. Fatty tumours are very rare. Small reddish calcareous nodules are sometimes found growing from the choroid plexus, and have received the name of psammomata.

CYSTIC FORMATIONS are found within the skull, being derived from several sources: as the result of hæmorrhage or softening; as in porencephaly; in connection with the degeneration of sarcomatous and gliomatous tumours; or they may be the results of parasites, hydatid or cysticercus, the former single, the latter multiple.

Brief reference may here be made to INTRACRANIAL ANEURYSMS. The larger arteries are those commonly affected. They are most frequently seen in the middle cerebral, and to an almost equal extent in the basilar; then follows in order of frequency the internal carotid, the anterior cerebral, the posterior and anterior communicating, and the vertebral arteries (Gowers).

The effects of intracranial new growth may be produced by the presence of a localised collection of pus within the brain, but this condition has been described under "Cerebral Abscess" (p. 88).

Symptomatology.—A new growth within the closed cranial cavity gives rise to certain phenomena by which its presence may be suggested. In the first place, if its size is considerable or its growth rapid, it may so interfere with the vascular and lympathic circulation as to cause marked disturbance of the intracranial

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pressure. Secondly, by its mere presence, more particularly in some localities than in others, it may exert pressure upon distant structures, and thus give rise to a series of phenomena which have to be distinguished from the effects of destruction or irritation of the tissue in the immediate proximity of the new growth. Thirdly, tumours in the neighbourhood of the third ventricle and quadrigeminal bodies, by their obstructive influence upon the flow of cerebrospinal fluid and the venous circulation in the veins of Galen, give rise to great increase of intracranial pressure, with distension of the lateral ventricles, thinning of the cerebral substance, and flattening of the convolutions. Fourthly, in most cases of intracranial tumour some degree of chronic leptomeningitis is apparent, the existence of which is detected by a matting of the membranes in the interpeduncular space.

Symptoms more or less common to all Intracranial Growths—*Headache.*—This is a frequent, but not an invariable, symptom of intracranial new growth. It may be either general or local, having not necessarily any direct relation to the situation of the tumour, though in some cases tenderness may be obtained on pressure over the seat of the growth. On the other hand, headache referred to one frontal region has assisted the diagnosis of tumour in the opposite cerebellar hemisphere. It is the most constant of the general symptoms of cerebral tumour. It is more pronounced with tumours of the posterior fossa and of the cerebellum, in which cases the pain may extend down the neck; it is less marked with the gliomata than with the tuberculous growths. The headache is chiefly due to increase of intracranial pressure and to irritation of the cerebral membranes.

Vomiting is also a common, but not a constant symptom. It is projectile in character, and unassociated with gastric disorders. It may accompany a severe attack of headache or a change in the position of the head. It is most frequent with cerebellar tumour.

Optic neuritis.—This is the most important of the general signs of intracranial new growth. It is not necessarily accompanied by blindness, or even defect of vision. Its presence is most significant, although its absence does not negative the existence of tumour. It is early in onset and intense in character in tumours of the cerebellum and quadrigeminal region; it is late and slight in cortical new growths. It is usually present at some time before death in the majority (about 80 per cent) of all cases. It is usually bilateral, and more marked on one side; but there is no constant relation between the intensity of the neuritis and the side of the lesion, though the

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more intense neuritis is often on the side of lesion. It tends to disappear after trephining. Should subsidence not occur atrophy of the optic nerve ensues.

Of the less common general symptoms may be mentioned—*vertigo*, which is more frequently associated with tumours of the cerebellum and its peduncles, though it is not uncommon in other intracranial conditions, *e.g.* atheroma of the cerebral blood-vessels; *general convulsions* of an epileptic character, and in many cases indistinguishable from true epilepsy, (the onset of epilepsy late in life should suggest the presence of a cerebral tumour); and various *mental alterations*. Those mental symptoms, which would seem to be more especially associated with tumour formation independently of its position, are impairment of memory, irritability, and mental hebetude. More rarely at an early stage hysterical phenomena may obscure the diagnosis; while acute mania and delusional insanity may be temporarily present.

Two signs, not altogether trustworthy, may be mentioned—an increase of dulness in the percussion note over meningeal and cortical tumours, and a rise of three to four degrees in the cranial surface temperature.

Focal symptoms special to each locality.—In most cases the existence of a cerebral tumour may be diagnosed from the general symptoms. In some no particular diagnosis as regards locality is possible; in others this is assured by the addition of focal or localising symptoms, which are referable either to irritation or to destruction of the several parts of the brain. Hence the localising symptoms indicate lesions of either an irritative or a destructive character. But there is no actual line of demarcation between them; lesions which are irritative in the early stages may become destructive in the later, and *vice versa*. The phenomena of irritation consist in convulsions, local or general, paræsthesia, and subjective sensory symptoms in the region of the special senses. The phenomena of destruction are motor palsies, anæsthesia, and abolition or defect of special sensation.

1. PREFRONTAL AREA.—By this term is meant the portion of the cerebral hemisphere lying between the ascending frontal sulcus and its linear continuation to the superior longitudinal fissure and the frontal pole, including both the convex and mesial aspects.

Tumours of this region may spring from the dura mater and compress the brain, or arise in the frontal lobe itself. They may present the following symptoms:—

(*a*) Cases in which the general symptoms only exist.

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(*b*) Cases presenting certain characteristic mental phenomena. Such are lethargy, a tendency to fall asleep, and stupor, a state of dementia eventually supervening. These symptoms are more particularly seen when both lobes are involved, and, according to some, when the orbital surface is affected. As regards the side of the brain implicated, examination of the sense of smell may show defect or loss on the side of the lesion. The situation of the headache is not constantly frontal, but is more commonly referred to this than to any other locality.

(*c*) Cases with extension of the tumour into the Rolandic area and the centrum ovale, giving rise to various local palsies or hemiplegia.

(*d*) Cases in which the symptoms resemble those of cerebellar tumour.

2. ROLANDIC AREA and its subcortical white matter.—This comprises the so-called motor region of the cortex. Tumours of this region are manifested in the early stages by localised convulsion if superficial, by monoplegia if subcortical. Thus there may exist spasm or monoplegia affecting the face, arm, or leg. Almost invariably spasm is followed by paralysis of the part or parts convulsed. The portion of the face or limb primarily convulsed indicates the situation of the growth. Extension of the tumour is indicated on the one hand by increase in the range of the convulsions or of the paralyses; on the other hand, a superficial tumour growing deeply gives rise to a permanent paralysis, while a deeply situated growth approaching the surface adds convulsions to the already existing paralysis. The knee-jerks and other tendon reflexes are increased on the paralysed side.

Should the tumour involve the third left frontal gyrus in right-handed persons motor aphasia will result, while the appreciation of written and spoken language remains intact.

3. THE ANGULAR REGION.—Destruction of the cortex of this region is productive of word-blindness, *i.e.* loss of the power of understanding printed or written language, and the power of reading aloud and of copying. This may or may not be associated with hemianopsia. On the other hand, should a tumour involve the subcortical white matter so as to interrupt the optic radiations and the association fibres from the occipital lobe, hemianopsia may be found along with a form of word-blindness, in which the patient is able to write spontaneously and from dictation, but is unable to read what he has himself written (subcortical alexia of Dejerine). If the destruction of this area be extensive, to these symptoms may be added hemianæsthesia, and perhaps hemiplegia from implication

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of subjacent structures. There is no clear evidence that this region contains a centre for ocular movements or for elevation of the upper eyelid.

4. OCCIPITAL LOBE.—Tumours of this region, whether cortical or medullary, produce homonymous hemianopsia, or blindness in the correlated retinal segments. This symptom arises from interference with the optic radiations or of the occipital cortex both upon the mesial and the external surface, the whole of which is connected with certain basal ganglia :—external geniculate body and pulvinar thalami. The earliest indication of implication of the cortical visual centre may be an affection of the most special of the visual sensations, viz. loss of the sense of colour in corresponding fields—*hemichromatopsia*. These symptoms, though recognisable by the physician, may be in the early stages entirely overlooked by the patient.

Corresponding to the convulsions seen in irritation of the Rolandic area, colour and visual auræ may be met. Such symptoms are indicative of irritation of the occipital cortex.

5. TEMPORAL LOBE.—The greater part of this lobe forms one of the so-called "latent" areas of the brain. Destruction of the hind end of the first temporal gyrus on the left side occasions word-deafness owing to destruction of the cortical centre for the reception of spoken language. Cortical deafness from tumour in this locality is rare. New growths involving the under and mesial surfaces, especially towards the tip of the lobe, may be indicated by sensory auræ of taste and smell, rarely by palsy of these senses. The presence of hemiplegia and hemianæsthesia along with the above symptoms point to the existence of large tumours extending deeply into the brain.

6. CORPUS CALLOSUM.—Tumours of this region are rare. The symptoms are those of a general nature, to which are added ingravescent hemiplegia first of one and then the other side. Mental symptoms—drowsiness and stupidity ending in dementia—are pronounced. The symptoms indicate a centrally placed tumour extending outwards.

7. The BASAL GANGLIA.—It is not clear whether the symptoms of new growth in this locality point to destruction of the corpus striatum and optic thalamus as such, or arise from pressure upon the adjacent internal capsule. If the growth lies anteriorly hemiplegia is present; if posteriorly hemianæsthesia and hemianopsia are found. In most cases the trio of symptoms indicates the site of the lesion (*vide* p. 126).

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8. THE QUADRIGEMINAL REGION. — Two associations of symptoms point to tumour of this region—ataxy of a cerebellar character and impairment in the movements of the eye-globes—there being no visual defect other than may be produced by the optic neuritis which coexists. In some cases tremors of an intentional character have been observed. These are probably due to extension of the growth forwards into the tegmental region so as to involve the superior cerebellar peduncle. The knee-jerks are usually lost.

Tumours of the crus cerebri, pons, and medulla oblongata are rare, and do not require a special description. Full reference to lesions in these localities is made later in the chapter on Focal Diagnosis (p. 129).

9. THE CEREBELLUM.—The general symptoms of tumour in this locality are more pronounced than in tumour of the cerebrum. The situation of the headache, which may be severe, is variable, being in some cases occipital, in others chiefly frontal. The symptoms indicative of cerebellar new growth are a tendency to fall to one or other side, and a staggering gait. There is no constant relation between the side to which the patient falls and the side of the tumour. If the middle peduncle be involved, however, it would appear as if rotation took place towards the side of the lesion. Cerebellar ataxy results from lesion, if extensive, of the lateral as well as of the middle lobe. Tremors of the arms are rare, and usually point to implication of the superior cerebellar peduncle. Weakness of a hemiplegic or monoplegic nature is observed in some cases; but whether this is due to lesion of the cerebellum as such, as stated by some, or is a pressure symptom as believed by others, remains a moot point. Both knee-jerks may be absent or both exaggerated, or one may be more marked than the other. Reasoning from experiment the more pronounced of the jerks is on the side of the lesion. Of most localising value are unilateral palsies of the cranial nerves, commonly the eighth and the fifth, more rarely the seventh and the sixth. These point to the side on which the tumour lies. Other symptoms occasionally seen in cerebellar tumour are retraction of the head, extension of the lower limbs, and weakness of the spinal musculature.

Diagnosis.—Three points present themselves for solution: Is there a tumour? if so, what is its locality? and what is the nature of the new growth? The first point receives its answer by an appreciation of the general symptoms already described. The second from the study of positive localising phenomena, while the absence of such signs may be of value in locating the growth in one of the so-called "latent" areas of the cortex. A monoplegia or

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hemiplegia of gradual onset is very suggestive of cerebral tumour, and so also is a general convulsion of epileptic nature occurring in adult life and followed by paralysis of a monoplegic or hemiplegic character. The production of local symptoms, as the result of pressure of a tumour in a distant locality, should not be forgotten. As regards the third point there are no direct means of distinguishing one form of new growth from another. Indirect evidence may be obtained from the locality of the tumour and from the age of the patient (*vide* table, p. 105).

It is necessary also to differentiate certain conditions closely analogous to those of intracranial new growth.

(1) Chronic Bright's disease with atheroma of the cerebral arteries. Here albuminuria, atheroma of the radial or other palpable vessel and albuminuric retinitis are usually sufficient to differentiate them; but in some cases of cerebellar tumour a condition of the macula lutea and surrounding tissue is not to be distinguished from the retinitis of renal cirrhosis.

(2) Cerebral syphilis. This is characterised by a history of infection at no distant date, by nocturnal headache, localised convulsions, a random association of palsies, and a tendency to relapse and remission of symptoms.

(3) The points of differential diagnosis between tumour, abscess, and tuberculous meningitis are given in the following table:—

Tumour.	Abscess.	Meningitis.
History indefinite.	Otorrhœa or other local suppurative condition present.	Tuberculous history or diathesis.
Onset gradual.	Onset usually abrupt.	Onset rapid.
Papillitis usually well marked.	Papillitis often absent or late.	Papillitis rare.
Monoplegia, hemiplegia, or localised convulsions, in definite order.	Focal symptoms indicative of cerebellum or temporal lobe.	Irregular palsies and convulsions.
Febrile symptoms absent.	Temperature sometimes subnormal.	Temperature irregular.
Duration months to years; regular course.	Duration variable with latent periods.	Duration of weeks, at times irregular.

In some rare cases at the onset it may be difficult to differentiate the symptoms of cerebral tumour from those of hysteria, chronic plumbism, and severe anæmia with optic neuritis.

Percussion of the skull has been attempted and recommended in the diagnosis of cerebral tumours. The normal cranial note is one of resonance, except in the aged, when it becomes considerably

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diminished. Before puberty the note is highly resonant. In how far a local decrease of resonance is to be regarded as symbolic of an underlying tumour is a point upon which there is no certain evidence. A diminished resonance has also been found over abscess and hæmorrhagic effusions.

Whether the application of the *X-rays* for purposes of focal cerebral diagnosis may be relied upon is also doubtful; should, however, a deepened shadow be present in a locality, destruction of which is known to produce certain phenomena, exhibited by the patient, strong corroborative evidence is thereby obtained. Taken by itself this is of little value.

Prognosis.—In the majority of cases the diagnosis of cerebral tumour, excepting gumma, is equivalent to a sentence of death. Some undoubtedly stop growing and shrivel, but the vast majority progress to a fatal termination. The duration of the symptoms varies widely, from a few months to several years. As a rule the symptoms steadily progress until coma supervenes, or sudden death terminates the scene. The latter is commoner in cases of cerebellar tumour. Surgical interference has to some extent mitigated the severity of the prognosis, as statistics show that recovery may reasonably be expected in 7 per cent of intracranial new growths, or even in 14 per cent according to the latest figures. There is no constant relation as regards the onset of general and local symptoms, sometimes the one, sometimes the other existing alone for months. The duration of the disease naturally varies from several weeks to a few months, although in exceptional cases the symptoms have been known to exist over some years.

Treatment.—The treatment of intracranial tumours resolves itself into the medicinal and the operative, the former consisting chiefly in the relief of symptoms. In all cases coming under treatment for the first time, whether there is a history of syphilitic infection or not, iodide of potassium in large doses should be prescribed, for, quite apart from lesions of a specific nature, it is often productive of much temporary benefit in tuberculous and sarcomatous new growths. Treatment by iodide should be maintained for two months, by the end of which time, should the growth be syphilitic, material improvement will have resulted.

As regards the relief of symptoms, headache is the one which will probably tax the therapeutic resources of the physician to the utmost. All the various analgesics in the market may be tried. Most of them will prove but of temporary service. Ice-bags to the head, blistering, and anodyne liniments may be used as adjuncts to

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the above or alone. A combination of butyl-chloral and gelsemium has been found of temporary value. Eventually morphia will have to be used should trephining not be performed.

Vomiting and giddiness may be relieved by the bromides, and insomnia by one or other of the numerous hypnotics.

Local application of electricity to paralysed limbs is of little value so long as the disease is active, but should arrest of the growth take place, its application will improve the nutritive condition of the muscles.

The operative treatment of intracranial tumour is undertaken with a twofold object, primarily for removing the disease should that be possible, and, failing that, of relieving the intracranial pressure, to the increase of which the general symptoms are largely due. In cases, therefore, in which there is no possibility of reaching the tumour, trephining is justifiable for the latter object alone.

From this it will be seen that the chief object of trephining in intracranial tumour is to relieve the more urgent and distressing symptoms. Of these headache is the one which is largely alleviated. The influence of a trephine opening upon the course of optic neuritis is important, as in some cases it has been found to resolve after opening the skull; destruction of the nerve should not have been allowed to advance too far before the operation is undertaken. A complication associated with opening the skull in cases of rapidly-growing tumours is hernia cerebri.

The tumours which are amenable to operation are those growing from the membranes and involving the cerebral cortex; those primarily implicating the cortex; those originating in the centrum ovale and growing towards and involving the cortex; and tumours of the lateral lobes of the cerebellum. The sarcomata being encapsulated growths are most satisfactorily removed, while the gliomata are unfavourable owing to their infiltrating and cystic nature. Tuberculous tumours are frequently multiple. A localisable gumma should be operated upon if resolution has not been complete under specific treatment within a reasonable period.

It was at one time thought that *lumbar puncture* might prove an effective means of lessening the increased intracranial pressure, which more especially accompanies tumours of the posterior cranial fossa. In some instances in which this was tried, no benefit was obtained, while in others it seemed as if the procedure was actually injurious.

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FOCAL DIAGNOSIS

There falls to be considered under this heading the symptomatology of lesions involving the several parts of the brain between the cerebral cortex and the decussation of the pyramids. The nature of the lesion only affects the localising symptoms in so far as it may occasion disease of sudden or slow onset, of rapid or pro-

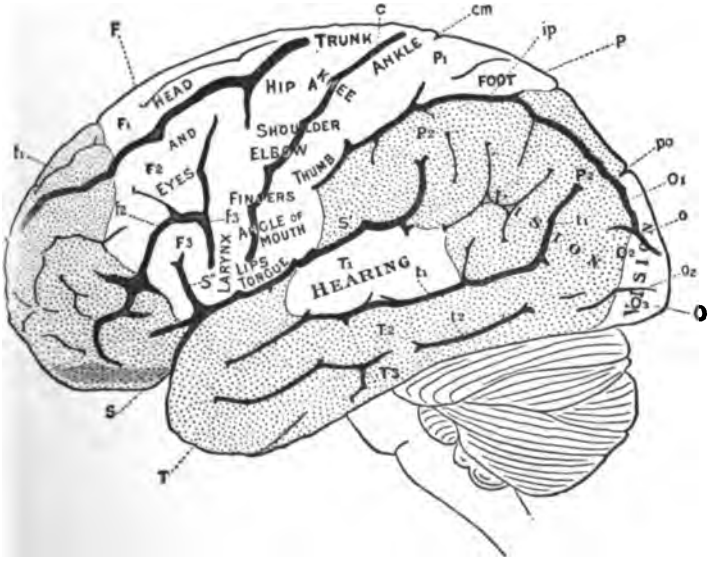

FIG. 8.—The external surface of the left cerebral hemisphere, showing the motor and sensory areas. The shaded portions correspond to Flechsig's "association" centres. (Modified from Ecker and Flechsig, by W. A. Turner.) The lettering indicates the lobes, gyri, and sulci. *F*, frontal lobe; F_1, F_2, F_3, the first, second, and third frontal gyri; f_1, f_2, superior and inferior frontal sulci; *T*, temporal lobe; T_1, T_2, T_3, the first, second, and third temporal gyri; t_1, t_2, the superior and inferior temporal sulci; *O*, occipital lobe; O_1, O_2, O_3, the first, second, and third occipital convolutions; *P*, parietal lobe; P_1, the superior parietal lobule; P_2, the inferior parietal lobule; *S*, the fissure of Sylvius; S', posterior limb; S'', ascending limb; *po*, parieto-occipital fissure; *ip*, the intra-parietal fissure; *C*, the fissure of Rolando. Motor areas are marked by small, sensory areas by large capital letters.

longed course, and of progressive or stationary nature. It is proposed to state in this connection those facts only which are generally accepted, giving special prominence to those verified by human pathology.

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For purposes of description the cerebral cortex is divided into certain areas, based not so much upon anatomical features and landmarks, as upon experimental and clinico-pathological data. In this way the cerebral cortical gray matter has been mapped into various well-known regions. These are the so-called "motor areas" lying around the fissure of Rolando, and the "sensory centres" which are depicted in the figures 8 and 9. Certain other regions

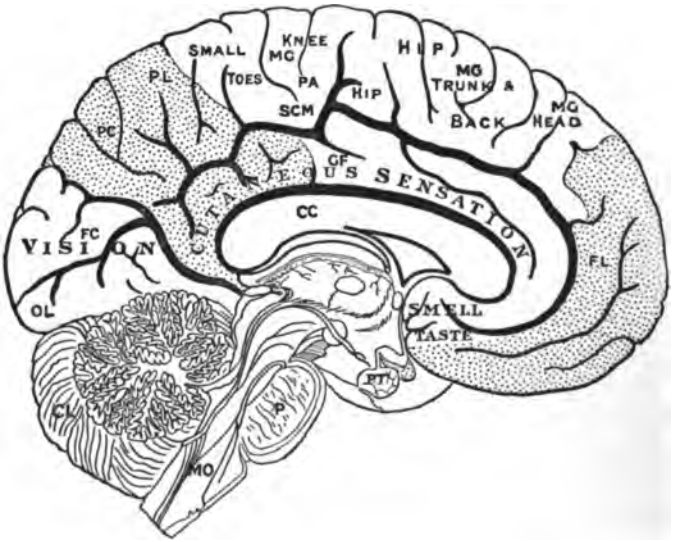

FIG. 9.—The mesial surface of the left cerebral hemisphere, showing the motor and sensory areas, and the "association" centres of Flechsig (shaded). (Modified as Fig. 2 by W. A. Turner.) *FL*, frontal lobe; *GF*, gyrus fornicatus; *MG*, marginal gyrus; *CC*, corpus callosum; *PL*, parietal lobe; *CL*, cerebellum; *PC*, precuneus; *MO*, medulla oblongata; *FC*, cuneus; *P*, pons; *OL*, occipital lobe; *SCM*, sulcus calloso-marginalis.

have received the term "latent" from the fact that their destruction causes no obvious impairment of function. To believe that such regions are "without function" is erroneous, inasmuch as the greater part of the cerebral cortex is connected with the basal structures by corticipetal fibres. Hence the so-called "latent" areas are probably concerned in the reception of unconscious impressions, or in the transmission of impulses to other parts of the brain-cortex. They form the "association centres" of Flechsig. (*Vide* also p. 31.)

Cortical lesions may be roughly divided into the irritative and the destructive; the former being characterised by convulsion or

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spasm, the latter by paralysis. A lesion, irritative in the first instance, may eventually produce paralysis by destruction of adjacent tissue and annihilation of its function. A lesion, primarily destructive, may assume an irritative character, from extension into tissues of an excitable nature, such as the cerebral cortex. It is also well known that some cerebral lesions may be entirely latent, their existence being only detected in the post-mortem room. Another and less well-defined influence exerted by a cerebral lesion, more especially of sudden onset, lies in the disturbance which may be occasioned in structures situated at some distance from the seat of disease. These effects are of an indirect nature, and may be the means of complicating diagnosis.

The Frontal Lobe

The posterior boundary of this lobe is the ascending frontal sulcus and its theoretical prolongation to the superior longitudinal fissure. The corticifugal tract from the anterior part passes through the anterior limb of the internal capsule, and the most mesial division of the pes cruris to the upper segment of the pons Varolii, while that from the posterior region lies behind and outside the former, and may be traced amongst the pyramidal fibres of the pons and medulla. The frontal corticipetal tract arises in the optic thalamus, and passes to the cortex of the frontal convolutions.

The cortex of the frontal lobe is supplied from two arterial sources, the whole of the mesial surface, along with the first and second convolutions of the convexity from the anterior cerebral artery, the remainder of the lobe consisting of the third frontal gyrus from a branch of the middle cerebral artery (Figs. 10 and 11).

Experimentally the frontal lobe, as above defined, may be subdivided into two areas—a prefrontal or non-excitable; and a post-frontal area, stimulation of which causes conjugate movement of the eyes to the opposite side. A destructive lesion is only productive of outward phenomena when the post-frontal area is affected, a temporary deviation of the head and eyes to the side of lesion being observed. No obvious motor or sensory result follows extirpation of the prefrontal lobe.

Unilateral extirpation of the whole frontal lobe is rarely followed by any symptom other than a temporary inability to turn the head and eyes to the side of the lesion, but bilateral extirpation is characterised by a series of mental phenomena closely resembling those resulting from progressive disease in man. As regards the ocular movements following bilateral removal, the general statement may be made that

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there is no impairment other than a temporary paralysis of conjugate movement.

Symptomatology.—Lesions of the frontal lobes may be entirely latent. As a rule, however, if of sufficient extent they are found to occasion a characteristic impairment of the mental faculties: failure of memory, a blurring of the power of attention, apathy, and a state of torpor, from which the patient may be aroused, but into which he speedily relapses, associated with which there is not unusually some degree of restlessness and irritability. These symptoms are said to be more pronounced when the orbital aspect of the frontal lobe is implicated. Only in rare instances have lesions of these lobes in man been productive of oculo-motor symptoms. As, however, in this locality disease is usually of a progressive nature, evidence of involvement of adjacent structures is forthcoming in paralysis of motion, and more rarely of sensation in a limb or limbs on the opposite side of the body. Unilateral loss of the sense of smell has been found of great localising value in tumours of the frontal lobe, this symptom arising from pressure upon the subjacent olfactory tract.

But there is one part of the frontal lobe which requires special reference—the third frontal convolution. Destruction of this region is followed by motor aphasia, on the left side in right-handed persons, and on the right side in the left-handed. As the artery which supplies this convolution—the inferior external branch of the middle cerebral—also supplies the adjacent part of the ascending frontal gyrus, motor aphasia resulting from thrombosis of this vessel is associated with paralysis of the tongue and angle of the mouth, or with a hemiplegia if the middle cerebral artery is itself obliterated. (See "Aphasia," p. 144.)

Some cases of tumour of the frontal lobe have been characterised by cerebellar symptoms; but this would appear to be due entirely to backward pressure upon the cerebellum from the anteriorly situated new growth.

The frontal lobes are frequently damaged in fracture of the base of the skull, more especially in those cases in which the blow or fall has been upon the occipital region or of the orbital plate. The symptoms resulting from the laceration thus produced are usually obscured by the general symptoms of cerebral compression.

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The Rolandic Area

This region comprises the convolutions bounding the fissure of Rolando, viz. the ascending frontal and ascending parietal gyri and the base of the superior frontal, the superior parietal lobule, and, on the mesial surface, the corresponding portions of the marginal convolution. The corticifugal tract is the great pyramidal system; while the corticipetal system consists of the corona radiata thalami, which is composed of the cerebral continuations, direct and indirect, of the fillet, superior cerebellar peduncle and the short tegmental tracts of the pons and medulla.

The *vascular supply* of the Rolandic area is, upon the convexity, the Sylvian branch of the middle cerebral artery, which also supplies the adjacent inferior parietal lobule and the first temporal convolution, and on the mesial aspect the anterior cerebral artery (Figs. 10 and 11).

Experimentally the Rolandic area is seen to be divided into several zones, electrical stimulation of which has been found to occasion movements of the limbs and body on the opposite side; thus, stimulation of the upper third gives rise to movements of the lower limb, the more proximal movements (hip) in the anterior part, the more distal (ankle, toes) behind the fissure of Rolando. In the middle third of this area the upper limb is represented, the proximal parts of the limb being above the more distal; while it is important to note that the thumb is especially represented at the anterior end of the intraparietal sulcus (Fig. 8). In the lower third the movements of the face, mouth, tongue, and larynx are represented, the face occupying the upper, the mouth and tongue the lower portion, while stimulation of the most anterior part of the zone produces movements of the vocal cords.

Excitation of the corresponding portions of the marginal gyrus is productive of movements of the limbs, but more especially of the body.

Conversely, experimental destruction brings about paralysis of movements of the limbs on the opposite side; if the whole area be destroyed hemiplegia results, if only in part, then monoplegia—leg, arm, or face, according as the upper, middle, or lower thirds are involved. Some movements are bilaterally represented, such for instance are those of the back, the hip, the shoulder girdle, the mouth, the tongue, and the larynx; therefore for complete paralysis of their movements, bilateral destruction of their centres is required.

Symptomatology.—Lesions of the Rolandic area give rise to clinical phenomena closely resembling those produced experimentally in the higher mammals. Hence there are irritative and destructive lesions causing convulsive and paralytic symptoms; the former if the lesion is situated superficially, the latter if subcortical.

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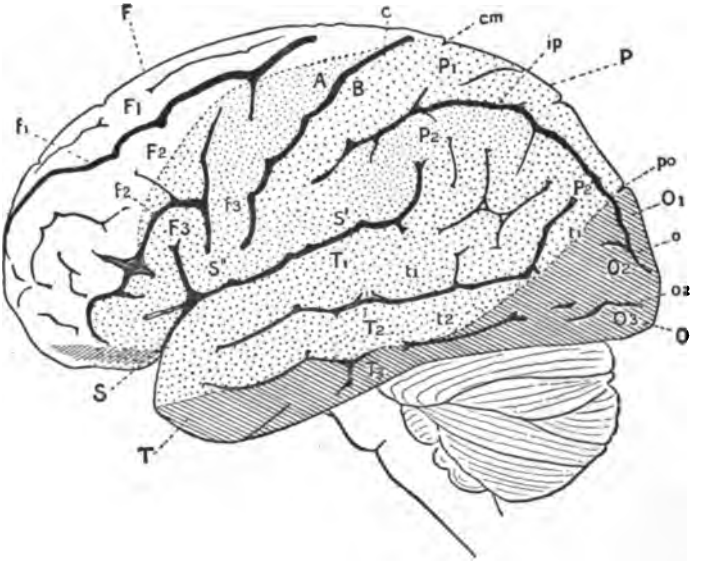

FIGS. 10 and 11.—The external and mesial surfaces of a cerebral hemisphere, showing the arterial supply of the cortical areas. (Modified from Bristowe.) Anterior cerebral artery = plain areas; middle cerebral artery = dotted areas; posterior cerebral artery = lined areas. The distribution of the middle cerebral artery is limited to the convexity of the brain. It divides into *five* branches: *a* supplies the inferior frontal gyrus; *b* supplies the ascending frontal gyrus; *c* supplies the superior parietal lobule; *d* supplies the angular gyrus and adjacent portions of the supra-marginal and first and second temporal convolutions; *e* supplies the anterior part of the temporal lobe.

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Convulsion starting in the lower limb points to an irritative lesion of the upper third of the Rolandic area, the part of the limb in which the convulsion starts indicating the portion of the area originally or chiefly affected. Similarly, a convulsion starting in the thumb, wrist, or forearm points to a lesion of the middle third of the Rolandic area; if in the face, lips, or tongue, to irritation of the lower end of the ascending frontal convolution.

Convulsions arising from cortical irritation are followed by temporary paralysis of the part or parts convulsed; hence a cortical irritative lesion may be temporarily succeeded by a cortical monoplegia. This condition is, however, more commonly due to subcortical extension, where a new growth, or other progressive lesion, in the fulness of time interrupts the pyramidal fibres in the centrum ovale. Just as there were three varieties of convulsion arising from cortical irritation, so there may be distinguished three chief varieties of monoplegia—the crural, the brachial, and the facial. The first indicates a lesion of the upper third, the second of the middle third, and the last of the lower third of the Rolandic subcortical white matter. Destructive lesions of the marginal convolution are rarer than those of the convexity, but the same general principles apply to them. The leg only is paralysed in affections limited to the paracentral lobule.

Combinations of the several forms are not uncommonly seen. Thus there may be a brachio-crural or a brachio-facial monospasm; as also a brachio-facial and brachio-crural paralysis. Paralysis which began as a crural monoplegia may, from the progressive nature of the lesion, eventually become a hemiplegia. Paralysis of one vocal cord (laryngoplegia) does not occur as the result of a unilateral cortical or subcortical lesion.

It is still a controversial point in how far destructive lesions of the Rolandic cortex directly occasion interference with cutaneous sensibility on the paralysed side. It would appear as if extensive cortical lesions were associated with hemianæsthesia, while limited destruction did not produce this result. This sensory defect is rarely as extensive as the motor palsy, being confined chiefly to the extremities of the paralysed limbs, and involving tactile sensation and the sense of position of the limbs rather than painful sensibility. On the other hand, sudden and extensive cortical lesions may occasion, in addition to hemiplegia, temporary anæsthesia over the whole of the opposite side of the body; but in these cases hemianopsia has also been observed, a fact which would point to an incidental relation between such lesions and the visual and sensory phenomena.

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The Gyrus Fornicatus

This is the convolution which bounds the corpus callosum and separates it from the marginal gyrus. Passing round the splenium it merges into the gyrus hippocampi, the two together forming the falciform lobe. The corticipetal fibres radiate out of the optic thalamus through both limbs of the internal capsule, along with the other afferent thalamo-cortical fibres. There does not seem to be a corticifugal tract from this region. The falciform lobe would appear to be connected with the Rolandic area by means of the fibres of the cingulum.

Experimentally destruction of the gyrus fornicatus has been found to give rise to abolition of cutaneous sensation on the opposite side of the body, painful as well as tactile sensation being affected. The anæsthesia thus produced is of a more persistent character than that following ablation of the Rolandic area. Owing to the anæsthetic state of the limbs a false appearance of paralysis is observed.

This convolution seems to be peculiarly free from the influence of morbid conditions, so that it is scarcely possible to formulate a definite symptomatology. In the few cases in which a stationary lesion has been found, cutaneous sensation was impaired or abolished; while in the cases of tumour there was in addition some motor weakness.

The Inferior Parietal Lobule

This lobule is the part of the parietal lobe of anatomists, which lies below the intraparietal fissure and is formed of two convolutions: the anterior or supramarginal gyrus, which lies immediately under the parietal eminence, and a posterior part or angular gyrus, which bends round the hind end of the Sylvian fissure, fusing in front with the supramarginal and below with the first temporal gyrus. Posteriorly it fuses with the annectant gyri. The connections of the angular gyrus are important; it does not appear to have a corticifugal tract, but it receives a large corticipetal system of fibres from the optic thalamus. The angular gyri are commissurally connected through the splenium corporis callosi. They form an important part of Flechsig's parietal association centre (Fig. 8).

The vascular supply of the inferior parietal lobule is from the Sylvian branch of the middle cerebral artery (Fig. 10).

The results of *experimental destruction* of this region are not in complete harmony, but it would seem to be certain that unilateral ablation of the angular gyrus is followed by temporary blindness of the opposite eye. Although electrical irritation of this lobe has been found

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to give rise to ocular movements, no oculo-motor paralysis follows its extirpation, even in cases in which the frontal lobes had been previously removed.

Symptomatology. — There is one characteristic symptom following destructive lesion of the angular gyrus in man, viz. word-blindness, or inability to appreciate the meaning of written language. This, however, would appear to be limited to lesions affecting the left side only.

Word-blindness occurring alone indicates a lesion limited to the cortical gray matter; in many cases, however, it is associated with hemianopsia, or blindness of the correlated retinal half-fields (see "Aphasia," p. 147). This association points to implication of the immediately subjacent optic radiation. There is some, but scarcely conclusive, evidence of a "crossed amblyopia," or defective vision of the opposite eye in cases of unilateral lesion of the angular gyrus.

There is no certain evidence that lesions of the inferior parietal lobule occasion oculomotor paralysis, or any defect in the elevation of the upper eyelid.

Except in the early stages of progressive, and in stationary lesions, the effects of destruction are rarely so limited as above described; in many cases hemiplegia and hemianæsthesia from extension into the centrum ovale or from pressure upon the internal capsule are observed.

The Occipital Lobe

This term includes the posterior end of the cerebrum, both upon its convex and mesial aspects. The anterior boundary is formed on the mesial surface by the internal parieto-occipital fissure, and on the convexity partly by the external limb of this fissure, the remainder fusing with the annectant convolutions. A corticifugal tract passes to the pulvinar thalami and the anterior quadrigeminal bodies, the corticipetal system consisting of the optic radiations, which terminate not only in the cuneus but also in the external occipital gyri and the lingual lobe. The occipital lobes are commissurally connected through the splenium and forceps corporis callosi.

The *vascular* supply is from a branch of the posterior cerebral artery, distributed both to the mesial and outer surfaces as well as to the white matter. This artery also supplies the ventral and mesial surfaces of the temporal lobe (Figs. 10, 11).

Experimental ablation of one occipital lobe gives rise to homonymous hemianopsy, or blindness of the half-fields to the opposite side. Similarly destructive lesion of this lobe in man is characterised by

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homonymous hemianopsia, the patient being blind to the side opposite the lesion. From numerous clinico-pathological observations, it would appear as if no particular region of the occipital lobe especially presided over this function, although one case has been recorded which limits the cortical centre for vision to the lips of the calcarine fissure (Henschen). Although it is probable that the several retinal segments have special cortical centres, there is no clear evidence as to their arrangement.

Symptomatology.—Hemianopsia following lesion of the occipital lobe may not be complete, that is to say, in place of blindness in the half-fields colour vision only may be abolished, or with the loss of the sense of colour there may be defect in form-sense. The view most in accordance with the facts, so far as yet ascertained, is that loss of colour vision in the half-fields, and hemianopsia, are merely manifestations of varying degrees of visual disturbance in the cerebral centres for sight; for it is known that hemiachromatopsia may pass into complete hemianopsia on extension of the lesion. On this hypothesis the loss of colour vision is regarded as the earliest manifestation of implication of the visual centres, and depending upon the extent and intensity of the destruction, more or less defect of form and light-senses is associated with it. No support can therefore be given to the opinion that centres exist for colour vision, apart from those for form and light.

Sector or *quadrant* hemianopsia, in which correlated retinal segments have been rendered blind, by lesion of the occipital lobe is occasionally observed. There is no certain evidence as to the representation of the several portions of the retinæ in the cortical visual centre; but it has been stated that the dorsal quadrants are represented in the upper part, and the ventral quadrants in the lower part of the occipital area (*vide* p. 29).

Homonymous hemianopsia existing by itself is characteristic of a destructive lesion of the occipital lobe on the opposite side. In many cases, owing to the progressive nature of the lesion, there is an associated hemiplegia or hemianæsthesia, from implication of centres and tracts in adjacent localities, such as the internal capsule (see also p. 127).

The Temporo-sphenoidal Lobe

This lobe occupies that portion of the cerebrum which lies below the fissure of Sylvius. It forms also a portion of the under surface of the hemisphere, and extends round on to the mesial aspect as far as the

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great transverse fissure. Its anterior end is prominent and free, its posterior fuses with the lower annectant gyri and the occipital lobe. The corticifugal tract passes from the first and second convolutions to the upper portions of the pons Varolii, while the corticipetal fibres form a part of the corona radiata thalami. A special radiation of corticipetal fibres may be traced chiefly to the first temporal convolution after destruction of the internal geniculate body, which tract would appear to form the cerebral segment of the central auditory system.

Its *vascular supply* is from two sources — the Sylvian branch of the middle cerebral artery supplying the first and second convolutions, and the posterior cerebral artery supplying the remainder of the lobe on the convexity, and the under surface.

Unilateral ablation is not characterised by any obvious symptom, but bilateral extirpation is followed by deafness (Ferrier).

Symptomatology. — There is one symptom which is pathognomonic of a destructive lesion, more especially of the first convolution on the left side, viz. word-deafness, or inability to comprehend spoken language. The localisation of this function on the left side is in harmony with the general left-sided nature of speech processes, both motor and sensory, in right-handed persons (see "Aphasia," p. 145). Whether a unilateral lesion in man is productive of deafness to ordinary sounds in the opposite ear is not certain, owing to the difficulty of eliminating aural complications; but there are a few well-authenticated cases of bilateral destruction of both superior temporal gyri causing complete deafness. In support also of the view that the first temporal gyrus in man contains the cortical centre of hearing, is the observation that irritative conditions of this region have caused subjective auditory sensations.

It would seem as if the anterior end of the temporal lobe upon the mesial surface—the uncinate gyrus—was specially devoted to the function of olfaction, both upon experimental grounds, and from the evidence of a few rare cases in which a tumour in this locality gave rise to subjective sensations of smell.

A large portion of the temporo-sphenoidal lobe may be regarded as "latent"; and for this reason destructive lesions of considerable size may exist without any localising symptoms at all. On the other hand, owing to pressure upon adjoining structures, more especially the posterior limb of the internal capsule, hemiplegia and hemianæsthesia may be produced.

The Corpus Callosum

The corpus callosum is the great commissure of the cerebrum, the

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majority of the fibres of which join corresponding portions of the cerebral hemispheres. It is also to a small extent a decussation of corticipetal fibres, which pass from the optic thalamus of one side to the opposite hemisphere. In the body of this structure the fibres are closely packed, but at the margins they ray out, and mingle with the other fibres of the centrum ovale.

The corpus callosum is rarely the seat of disease. A few cases of tumour have however been studied and recorded, and from these the following have been stated to be characteristic symptoms: the gradual incidence of hemiplegia, first on one side, and then on the other, associated with stupidity and lethargy, and a progressive dementia ending in coma. There is usually also a freedom from the general signs of intracranial new growth, headache, sickness, and convulsions.

The Internal Capsule and Basal Ganglia

The internal capsule is a strand of white fibres separating the nucleus caudatus and optic thalamus, which lie on its mesial aspect, from the lenticular nucleus which forms its outer margin. It is subdivided into an anterior and a posterior limb, the angle formed by these two constituting the knee. In both limbs are to be found fibres ascending to, and descending from, the cerebral cortex. In the fore part of the anterior limb are found the corticifugal and corticipetal fibres of the frontal lobe. Immediately behind them are the pyramidal fibres for the conjugate movement of the eyes; at the knee are the fibres for the mouth and tongue—the so-called geniculate fasciculus—and then follow those for the face, the upper limb, the trunk, and the lower limb occupying the first two-thirds of the posterior limb of the capsule. Behind this comes the combined sensory tract, extending as far back as the posterior extremity of the lenticular nucleus. Occupying the retro-lenticular part of the capsule is the optic radiation. It is not quite clear where the fibres serving taste and smell lie, but that they are in close relation with the other sensory fibres is evident from the fact that these senses may be affected by lesions of the posterior part of the capsule. The sensory auditory tract passes from the internal geniculate body through the retro-lenticular part of the capsule.

The *vascular supply* of this region is simple. The anterior limb of the capsule and the nucleus caudatus are supplied by the lenticulo-striate artery, the posterior limb and lenticular body by the lenticulo-optic artery, and the pulvinar thalami by the external optic artery. Owing to the freedom with which these rupture, the term "arteries of cerebral hæmorrhage" has been applied to them.

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Symptomatology.—From the arrangement of the motor fibres in the internal capsule already given, it is clear that a lesion of the anterior two-thirds of the posterior limb gives rise to hemiplegia of the opposite side. Should the lesion involve the hinder part of the motor tract, the leg will be chiefly affected, and with this type there is commonly hemianæsthesia. If accompanied by homonymous hemianopsia the lesion extends into the

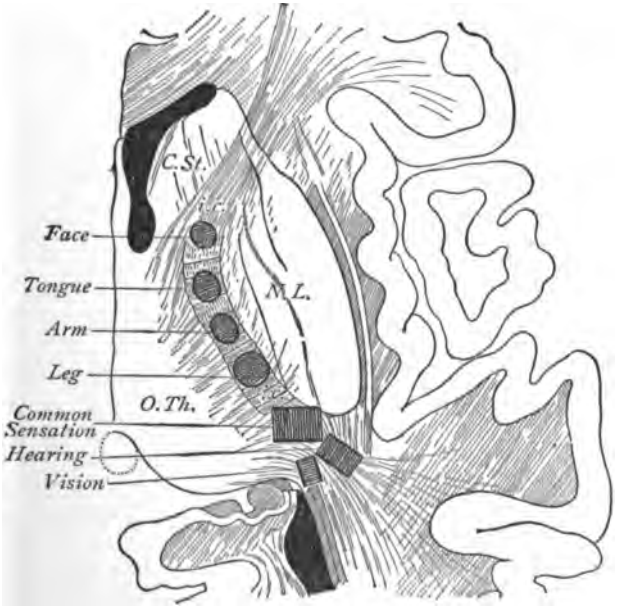

FIG. 12.—The internal capsule, showing the position of the afferent and efferent tracts. (Modified from von Monakow.) The circles represent the bundles of motor (pyramidal) fibres, the squares those of special sensation. *C. St.*, corpus striatum; *i. c.*, internal capsule; *N. L.*, nucleus lenticularis; *O. Th.*, optic thalamus.

retro-lenticular part of the capsule. Hence the triad, hemiplegia, hemianæsthesia, and hemianopsia, is characteristic of destructive lesion of the posterior limb of the internal capsule. Monoplegias from limited lesion of the internal capsule are theoretically possible, but are rarely seen as a result of this lesion; much more commonly are they due to a circumscribed lesion of the centrum ovale before the pyramidal fibres approximate so closely as in the capsule, or to interruption of the pyramidal fibres in the subcortical white matter.

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Of the basal ganglia, corpus striatum, and optic thalamus, the latter is the more important when viewed clinically. It is difficult to eliminate the effects of experimental irritation or destruction by disease of the corpus striatum from those of indirect implication of the adjacent capsular fibres; but the optic thalamus, and more especially the pulvinar, may be experimentally destroyed without injury to neighbouring structures (*vide* also p. 23). Such experimental destruction of the optic thalamus was followed by crossed amblyopia and hemianopsia and hemianæsthesia of the opposite side. That these symptoms were due to lesion of the ganglion was obvious from the intact condition of the adjacent structures. It was clear also from the study of secondary degeneration that a large number of ascending tracts, including probably those of common sensation, enter the optic thalamus before being transmitted to the cortex. Hence the symptoms which have been found to follow destruction of the optic thalamus by disease may be due to direct interference with the functions of this ganglion. On the other hand, it is clearly difficult to exclude some implication, either direct or indirect, of the adjacent internal capsule. From this the symptoms usually described as a result of lesion of this ganglion are hemiplegia, hemianæsthesia, and hemianopsia.

There are certain other phenomena, the causation of which is commonly ascribed to lesion of the optic thalamus. Such are abolition of the movements of emotional expression, as in laughing and crying, with retention of voluntary power over the face on the same side; and athetosis or mobile spasm of the paralysed arm. This latter is sometimes seen in adult hemiplegics, but more commonly in that arising in infancy and childhood. The so-called post-hemiplegic chorea has been ascribed to lesion of the optic thalamus. There is no certain evidence that the optic thalamus has any direct influence in the production of pyrexia.

Brief reference may be made here to the *hemiopic pupillary reaction*. By this condition is meant the contraction of the pupil which occurs when a pencil of light is thrown upon the seeing halves, and its absence when thrown upon the blind halves of the retina in cases of homonymous hemianopsia. When it is present the symptom indicates interference with the conduction of impressions along the optic tract. Should it occur with lesions obviously in other localities, its existence signifies pressure upon the optic tract.

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Corpora Quadrigemina

Of the four tubercles the two anterior have entirely different associations and connections from the posterior. The anterior receive fibres from the optic tracts, the occipital lobes, and the mesial fillet; they stand in indirect relation to the visual system. The posterior bodies receive chiefly the lateral fillet, and appear to be ganglia accessory to the auditory system.

In how far *experimental* destruction of these ganglia is followed by definite symptoms is a point upon which the evidence is contradictory, the older views that their destruction was followed by blindness and disturbances of station and locomotion not having received confirmation in recent experiments upon the higher mammals. It would now appear as if the effects of their ablation were largely of a negative character. The relation of the anterior bodies to the visual system is in the sphere of pupillary and ocular movements rather than in that of vision proper; while there is no clear evidence that destruction of the posterior bodies alone gives rise to impairment of hearing.

Symptomatology. — These ganglia are rarely the seat of disease other than tumour formation, and it is from cases of this nature that the symptomatology has been obtained. The two most characteristic symptoms are a reeling gait associated with impaired movement of the eyes. The gait is like that of a drunken person, being comparable to that of cerebellar disease rather than locomotor ataxy. In advanced cases the disorder of equilibration is so great that the patient is unable to stand. Even in the later stages there is no true paralysis, unless from extension of the disease the cerebral peduncles become involved. The oculomotor palsy consists rather of interference with the functions of the third and fourth nerves than of the sixth. Hence paralysis of convergence, ptosis, and loss of the upward movement of the globes are the chief phenomena. Vision is not materially altered, except in so far as the fundus oculi is locally affected, by neuritis or other indications of increased intracranial pressure. The difficulty of testing audition in these cases is great, owing to the mental apathy and listlessness usually present. It would seem, however, from a view of the cases in which this was carefully tested, that hearing was impaired on the side opposite the lesion, or in bilateral cases on the side opposite that which was most affected. Tremors, chiefly of the arms on voluntary movement, have been observed, and are similar to those met with in cases of disseminated sclerosis. They are probably due to an extension of the growth into the

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tegmentum and optic thalamus. The knee-jerks are as a rule exaggerated, but they may be abolished even at an early date.

It is convenient to describe in connection with lesions of the quadrigeminal bodies the condition known as *ophthalmoplegia*, or paralysis of the external ocular muscles from lesions in or about the nuclei in the gray matter of the Sylvian aqueduct. The chief form is ophthalmoplegia externa, in which the external ocular muscles are wholly or in part affected. The typical form is that arising from chronic nuclear degeneration, existing alone for a number of years, or associated with other chronic processes, such as bulbar paralysis or tabes dorsalis. Both eyes are usually affected, but the internal ocular muscles and the pupillary light reflex are rarely involved. Weakness of the orbicularis palpebrarum has been observed in some cases.

Another form of ophthalmoplegia, differing in its mode of onset and in its pathological characters from the above, is the so-called *polioencephalitis superior* (see p. 85). With this is found an ophthalmoplegia of sudden onset, and not uncommonly associated with general symptoms, such as headache, pyrexia, and optic neuritis. These cases are found to be due to a hæmorrhagic condition of the Sylvian gray matter and to be associated with chronic alcoholism or other toxic agents. There are also other but less severe cases of acute ocular palsy of central origin. These latter are not rapidly fatal, and in many cases recovery takes place. In them the causal agent is to be found in some blood poison, such as syphilis, diphtheria, influenza, and others as yet undetermined.

Various forms of partial ophthalmoplegia localisable to the same neighbourhood are not uncommon. Such are cycloplegia, or palsy of accommodation, iridoplegia or loss of the pupillary light reflex, paralysis of convergence, and ophthalmoplegia interna, in which all the internal ocular muscular mechanisms are paralysed, either on one or both sides.

From this it is evident that the presence of ophthalmoplegia, partial or complete, acute or chronic, which may be ascribed to disease of the central nervous axis, localises a lesion, whether degenerative or vascular, in the gray matter around the aqueduct of Sylvius and ventral to the quadrigeminal bodies.

Crura Cerebri

The crura cerebri connect the cerebrum with the pons Varolii, and are the means of transmitting afferent and efferent impressions to and

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from the higher regions of the brain. In the ventral part of each crus lie from within outwards the fronto-pontine, pyramidal and temporo-pontine tracts, while in the dorsal part or tegmentum are to be found the mesial fillet, the superior cerebellar peduncle, the central auditory tract, and the short fibre systems of the tegmentum, which last probably convey impressions of ordinary cutaneous sensation.

Symptomatology.—From the above it will be apparent that the symptoms following an extensive lesion of the crus may be numerous. The association of symptoms which characterise a lesion in this locality are paralysis of one side of the body accompanied by palsy of the third cranial nerve upon the opposite side, one of the series of alternate paralyses. This arises from the anatomical position of the third cranial nerve, which, as it passes from its nucleus to the periphery, penetrates the mesial portion of the crus. Owing to the juxtaposition of the sensory tract and the superior cerebellar peduncle, some amount of anæsthesia and intentional tremor may be observed on the hemiplegic side if these structures are implicated.

The lesions in this situation may be either intra- or extra-peduncular, the latter being the more common. In this there is an entire absence of those symptoms which point to implication of the tegmentum cruris. Paralysis of the third nerve existing upon the same side as the hemiplegia points to a double lesion, usually of the nature of a basal meningitis (syphilitic or tuberculous).

The Cerebellum

The cerebellum is connected with the central nervous axis by means of three peduncles: the superior, which transmits cerebellar efferent fibres directly to the optic thalamus and thence indirectly to the opposite cerebral hemisphere; the middle, which contains efferent fibres terminating in the opposite nucleus pontis; and the inferior, which contains the cerebellar prolongation of the posterior columns of the spinal cord of the same side (afferent) as well as the decussating cerebello-olivary efferent system. There is another and highly important connection between the middle lobe and the nucleus of Deiters, through which the cerebellum exerts an uncrossed influence upon the spinal cord by means of the tract which passes from this nucleus to the anterior horns through the antero-lateral descending spinal system.

Connections with the third and sixth nerve nuclei are also described.

The chief symptoms following *experimental* ablation of the cerebellum are highly characteristic. Immediately after removal the disturbance of equilibrium is so profound that the animal is unable to stand or

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walk; but in a short time some degree of stability returns, but the animal is easily upset by excited, or undue hurriedness of, movement. The gait is sprawling, there is marked unsteadiness of the limbs, body, and head, and on volitional effort tremors, having the character of those seen in disseminated sclerosis in man, are observed. The motor power is in no way affected except in so far as the tremors may interfere with sustained effort. If a lateral lobe only is removed, the symptoms are confined to the limbs on the side of the lesion; there is a tendency to fall to the side of lesion, the knee-jerk is brisker on that side, and in walking the animal inclines to deviate towards the side of extirpation (*vide* p. 20).

Symptomatology.—The chief symptom of cerebellar disease is a disturbance of the equilibrium, giving rise to the so-called cerebellar ataxy. The gait is reeling in character, resembling that of a drunken person rather than that of a tabetic. There is usually a tendency to fall to one or other side, but there is no certain relation between the side of the lesion and that to which the patient falls. Should there exist a constant tendency to rotate in one direction, this symptom points to interference with one of the cerebellar peduncles, and more especially the middle peduncle. In other cases the disturbance of equilibrium is so pronounced that the patient is unable to stand or even sit without support. This points more particularly to an implication of the middle, rather than of the lateral lobe. Recent investigation, however, shows that cerebellar ataxy is not peculiar to lesions of the middle lobe, as it occurs with lesions of the lateral lobe in cases in which the middle lobe is quite unaffected. Lesions of the middle lobe are more commonly associated with retraction of the head, arching of the back, and rigidity of the limbs.

In a certain percentage of cases of cerebellar tumour, no symptoms of a characteristic nature are detected. The explanation of this is to be found largely in the ability which the remaining portions of the cerebellum and the higher cerebral (motor) centres have of compensation, as the absence of phenomena is found chiefly in slowly progressive or stationary cases.

Cerebellar symptoms are invariably present in disease of sudden onset and of rapidly progressive tendency; while destructive lesions of experimental nature show the most characteristic effects of interference with the functions of this organ.

Tremors and irregular movements of the head, body, and limbs are rarely seen in cerebellar disease in man. If present in the limbs they are upon the side of the lesion, and are of an intentional

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character. Considering the constancy of their presence after experimental lesions, their rarity in man is notable.

In some cases weakness of the limbs upon the side of the lesion has been observed, in others upon the opposite side. True motor paralysis occurring in cerebellar disease would appear to be an indirect or pressure symptom, the apparent weakness observed in experimental cases being consequent upon the instability of the limbs, which is so constant a feature.

The state of the knee-jerks is uncertain. In some cases there is no jerk, in others both are exaggerated, while in others again one is present and the other absent or diminished. The more active of the two jerks is usually upon the side of the lesion. If there co-exists hemiparesis the jerk is exaggerated upon the paretic side.

Both nystagmus and deviation of the optic axes are observed in cases of cerebellar disease, but there is no constant relation between the direction of deviation and the side of the cerebellar lesion. In some cases coarse nystagmoid jerkings are seen on looking to one side, and fine movements on looking to the other. The lesion is more probably upon that side on looking to which the coarse movements are detected.

Symptoms of more localising value than any of the foregoing are seen in associated cranial nerve paralyses. Such are paralysis of the sixth nerve, and of the eighth nerve indicated by deafness. Palsy of the fifth nerve, whether motor or sensory, localises the lesion in the middle peduncle. Cranial nerve paralyses are upon the side of the lesion.

The following localising symptoms would therefore indicate the presence of a tumour implicating the right cerebellar hemisphere and middle peduncle: deafness in the right ear, unassociated with middle ear complications; an unsteady and uncertain gait, with a tendency to fall more particularly to the right side; coarse nystagmoid oscillations on looking to the right; movements resembling those of disseminated sclerosis on volitional effort of the right arm; an awkward and uncertain action of the right leg; a slight increase of the right knee-jerk; and perhaps slight blunting of sensibility over the right cornea and side of the face.

Pons Varolii

It may be briefly stated that the pons transmits motor impulses from and sensory impressions to the cerebrum; that in it are situated the nuclei of certain motor cranial nerves (fifth, sixth, seventh); that

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into it pass the roots of two sensory cranial nerves (fifth and eighth); and that it has an intimate association with the cerebellum through the middle peduncle. The pons receives its vascular supply mainly through the basilar artery and its branches (*vide* also p. 19).

For purposes of localisation the pons may be divided into a dorsal, or tegmental, and a ventral, or crustal, portion. Lesions of the former are manifested by affections of sensation, or of paralysis of cranial nerves, or by a combination of these; lesions of the latter by paralysis of a hemiplegic or diplegic character. Or again, lesions of the pons may be described according as they are situated, proximal or distal to the nucleus of the sixth cranial nerve. In the pons the nature of the lesion has an important bearing upon the symptoms, that is to say, whether it is of a degenerative or a gross character.

Symptomatology.—The essential features of pontine lesions are to be found in the so-called alternate paralyses. Of these there exist several types.

1. Paralysis of the conjugate movement of the eyeballs to one side with palsy of the opposite limbs.

2. Paralysis of the face of the peripheral type on one side and of the limbs on the opposite side.

3. Paralysis of conjugate movement and of the face on one side and of the limbs on the opposite side.

4. Paralysis of the fifth nerve, motor or sensory or both, on one side, and of the face (cerebral type) and the limbs on the opposite side.

5. Paralysis of one or more cranial nerves on one side, with palsy of motion or sensation or both on the other side.

But all lesions of the pons are not necessarily characterised by symptoms of this nature. A unilateral lesion in the upper part, proximal to the sixth nucleus, involving the crustal portion, may occasion an ordinary hemiplegic paralysis of the cerebral type; or the cranial nerves, one or more, may be affected without any limb palsy if the lesion is purely tegmental in situation.

Somewhat similar associations of paralyses may result from disease outside the pons in the subdural space, but if paralysis of the conjugate movements of the eyes is present, either alone or in combination with palsy of the seventh nerve, the lesion is of coarse nature and situated in the tegmentum pontis.

Lesions of a bilateral or multiple character, chiefly of the nature of small hæmorrhages or softenings, give rise to symptoms of a pseudobulbar type; such are weakness of the limbs on both sides, paresis of articulation and deglutition from interference with the

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movements of the lips, tongue, and soft palate, but rarely with any affection of common sensation. Various odd associations of palsies are highly characteristic of pontine lesions. Such, for instance, are paresis of three limbs, or a bilateral paralysis in which the limbs on one side are affected earlier than those on the other.

Affections of hearing are variable in pontine disease. Many cases are recorded in which deafness on one or both sides has been observed, but in others this symptom was entirely absent. The fact that the central auditory tract occupies a ventral and external position in the pons may explain its escape in many cases. Should the disease be situated outside the nervous axis, hearing may be impaired through implication of the auditory nerve itself.

Ataxic movements, more especially of the arms, have been described in disease of the pons, but it is not quite clear whether these are due to lesion of the mesial fillet in the pons or are pressure symptoms.

The Medulla Oblongata

Like the pons the medulla oblongata is both a transmitter of afferent and efferent cerebral impressions, as well as a centre of origin of some cranial nerves. It emits the hypoglossal and the motor fibres of the vago-glossopharyngeal nerves, and it receives the afferent fibres contained in the latter. It is also closely related to the cerebellum through the restiform body or inferior peduncle. At its lower end, where it fuses with the spinal cord, an important rearrangement both of the descending pyramidal and of the ascending posterior columnar fibres takes place. It may also be divided into a dorsal tegmental and a ventral pyramidal part.

As regards the **symptomatology** of disease of this locality little need be said, as the coarser forms of disease prove so commonly fatal. The chief interest lies in the study of chronic *progressive bulbar paralysis*. This condition is characterised by a gradual impairment in the movement of the lips, the tongue, the soft palate, the vocal cords, and the pharynx, so that articulation and deglutition become impaired. As the disease is part of a progressive affection of the motor neuronic systems, other structures become affected, such as the anterior horns of the spinal cord, the seventh and motor fifth cranial nuclei, and, in some instances, the oculomotor nuclei. As the disease advances speech is reduced to an inarticulate sound, the tongue cannot be protruded beyond the dental arch, saliva dribbles from the mouth, and swallowing is a matter of extreme difficulty. Just

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as in the analogous condition of ophthalmoplegia, bulbar paralysis may occur suddenly, sometimes the result of thrombosis of the bulbar arterioles, or from an acute inflammatory condition—*polio-encephalitis inferior*. This acute bulbar paralysis presents features anatomically similar to those of the chronic variety.

A form of alternate paralysis has been described as characteristic of coarse disease of the medulla, viz. paralysis of the tongue on one side and of the limbs on the opposite side.

Lesions in the neighbourhood of the pyramidal decussation, though rare, have a characteristic symptomatology. This consists of anæsthesia over the distribution of the fifth cranial nerve on the side of the lesion, with paralysis of the limbs on the same side, and analgesia of the body and limbs on the opposite side, an association which has been experimentally found to be due to destruction of the tubercle of Rolando.

WILLIAM ALDREN TURNER.

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APHASIA AND OTHER DEFECTS OF SPEECH

A proper conception of the physiological mechanism of speech is perhaps most easily arrived at by a consideration of the way in which speech is acquired by the infant in the process of its development. Within a short time after birth the child begins to recognise the nature and uses of some of the objects in the world around it, and to express its simple conscious processes by gestures. Long before it is able to utter any articulate sound the infant learns to connect certain sounds which it hears with certain objects, and these auditory word memories first implanted serve by far the most important function, as will be subsequently seen, in the processes and expressions of thought. That part of the nervous system where such memories are located is termed the "auditory word centre." Later on, guided by the auditory word memories, the child begins to express itself in articulate speech.

For the consideration of the nervous arrangements and processes in speech it is essential to bear in mind that all motor processes have been evolved from reflex actions, and that all living motion is sensori-originated, sense-guided, and sense-governed, and that a motor process of itself has no conscious concomitant, our consciousness of motor processes being the consciousness of the sensations which accompany the movement. When any act is learned the sensations accompanying the movement become implanted in the nervous system as "kinæsthetic memories"—the memories of the sensations accompanying movement. The portions of the nervous system where these memories are registered are termed "kinæsthetic centres," and it is from these centres that the incitations for motor processes proceed.

When learning to speak, memories of the sensations accompanying the movements of the articulatory and phonatory apparatus become implanted in a "kinæsthetic centre," that part of the nervous system where the kinæsthetic memories of the articulation and phonation of words are located being termed the "glosso-kinæsthetic centre." This centre, educated by the auditory word centre, remains subservient to it, and depends upon it for its incitation.

When the child learns to read, certain symbols (words) become connected with certain objects and ideas, and the memory of the word seen (visual word memory) becomes implanted in the "visual word centre." When he is learning to read, the meaning of the word seen is learned by the child from the meaning of the word which he hears spoken, and the auditory word centre, which at this period is well educated, serves as the instructor of the visual word centre. From this

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intimate original connection between these two leading speech centres it will be easy to understand that they are functionally dependent as leader and lieutenant centre for the recall of words in silent thought and for the production of speech, the auditory word centre being the chief.

Just as when articulation is learnt the glosso-kinæsthetic centre (the so-called motor vocal speech centre) is developed in connection with the auditory word centre, so when writing is learnt the memories of the sensations accompanying the movements of the hand in writing are registered in a kinæsthetic centre in connection with, and subservient to, the visual word centre. This centre is termed the "cheiro-kinæsthetic centre," and from this centre the incitations for the movements of writing proceed. The two kinæsthetic centres for speech in their turn are in connection with the motor centres respectively for the movements of articulation and phonation, and for the movements of the hand in writing.

It was long ago argued by Sir W. Broadbent that the four centres above mentioned, auditory and visual word centres, glosso- and cheiro-kinæsthetic, were governed by a single higher one, to which the term "ideational centre" was applied. In this it was supposed that the concepts for speech were elaborated and that impressions received by the lower speech centres were here combined, selected, and redistributed to the lower speech centres. Lichtheim subsequently strongly supported this view. Dr. Bastian, however, opposes the theory and his opinion is at the present time very generally followed. There is no pathological nor clinical evidence of the existence of such a centre. Lichtheim indeed drew from theoretical grounds the clinical pictures that should result from the isolated destruction of the ideational centre and of the paths connecting it with the lower centres. The rare cases conforming to his types, which have been subjected to pathological investigation, have invariably shown partial lesions in the auditory or visual word centre. Further, the theory of the ideational centre entails a centre for consciousness apart from the general sensory centres, while pathological and psychological evidence all goes to prove that consciousness has its seat in the interacting general sensory centres.

Situation and connections of the speech centres.—The four chief speech centres which have been above indicated are situated in the cortical gray matter of the left cerebral hemisphere. In the majority of left-handed persons, however, these centres are situated in the right cerebral hemisphere. They are specialised portions of the general centres for hearing, vision, movements of tongue, lips, etc., and movements of the hand respectively; and they are situated on the confines of such centres. Thus the auditory and visual word centres are situated at the limits of regions known to be connected with hearing and vision respectively. Sir W. Broadbent first pointed out, and Flechsig subsequently thoroughly investigated certain areas in the brain

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which never acquire direct connections with the basal ganglia or pyramidal systems, and which develop their medullated fibres long after birth, such fibres proceeding to the general centres of the cortex as part of the association and tangential systems. They have been termed the "sensory annexes," and are situated (1) in the posterior part of the parietal region; (2) in the upper part of the temporo-sphenoidal lobe; (3) in the interior region of the insula; (4) in the prefrontal lobe. The situation of the visual word centre corresponds with the first of these areas and that of the auditory word centre with the second. *The auditory word centre* occupies the posterior two-thirds of the first temporal convolution. *The visual word centre* is situated in the angular gyrus and in the adjoining part of the supra-marginal convolution, the two leading speech centres being thus in close proximity to one another around the hinder end of the fissure of Sylvius.

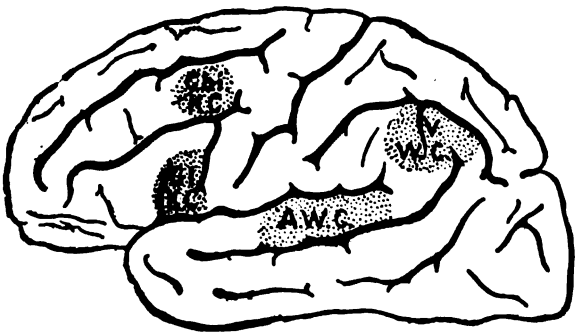

FIG. 13.—Diagram showing the approximate sites of the four word centres (Bastian).

The glosso-kinæsthetic centre is located in the posterior part of the third frontal convolution, extending slightly on to the foot of the ascending frontal gyrus (Broca's region). *The cheiro-kinæsthetic centre* is believed to be in the posterior part of the second frontal convolution (see Fig. 13).

In accordance with the developmental relations and functional dependence of these centres they come to be connected by *commissural fibres;* thus the auditory and visual word centres are intimately connected by fibres, one set of which conducts impulses from auditory to visual, and another set conducting visual to auditory word centres. A similar double set of commissural fibres binds the auditory word centre to the glosso-kinæsthetic centre, and another group unites the visual word centre and the cheiro-kinæsthetic centre. In certain highly educated persons, and also as a compensatory process for the destruction of certain parts of the speech mechanism by disease, connections

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other than these just described may be found. The usual and unusual parts of connection between the speech centres are clearly shown in Dr. Bastian's diagram[1] (Fig. 14).

The executive centres for articulate speech are situated in the medulla in the gray matter of the floor of the fourth ventricle (nucleus of hypoglossal nerves, motor nuclei of vagus group, etc.), and they are connected with the glosso-kinæsthetic centre by a band of fibres of the pyramidal system, which, in its downward path, occupies a position at the genu of the left internal capsule, and is called the "geniculate fascicle."

The executive centres for writing are situated in the anterior horns of the cervical enlargement of the spinal cord, and are connected with the cheiro-kinæsthetic centre by fibres of the pyramidal system.

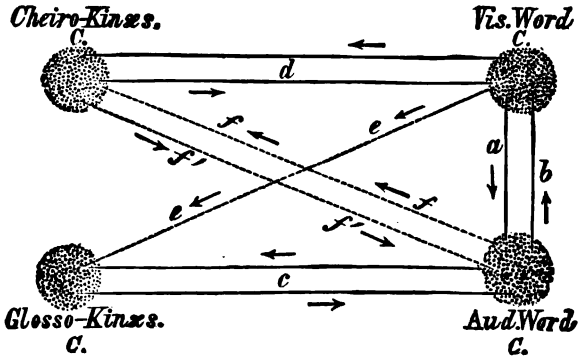

FIG. 14.—A diagram illustrating the relative positions of the different word centres and the mode in which they are connected by commissures. The connections indicated by dotted lines indicate possible but less habitual routes for the passage of stimuli (Bastian).

The location of the speech centres in the left cerebral hemisphere requires some explanation, since the right and left cerebral hemispheres are in equivalent positions on the receptive side. It is probable that, owing to the major use of the right hand in ancestors, the left cerebral hemisphere stands endowed at birth with a higher receptive capacity than does the right hemisphere, and since each degree of education will increase the receptive capacity, it follows that the right hemisphere will remain little educated in certain of the highest cerebral functions.

There are, however, in the right cerebral hemispheres speech centres of a similar nature and location to those in the left, but entirely subordinate to the latter, the two being intimately connected by commissural fibres of the corpus callosum. These centres are of great importance

[1] *Aphasia and other Speech Defects.* London, 1898.

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in the recovery of speech when the chief centres in the left cerebral hemisphere are destroyed by disease, and their capacity for taking over the function of such damaged centres seems to be much greater in childhood, and to decrease with advancing age. Thus the fact is explained that if destruction of the leading speech centres occur before the age of six years, permanent loss of speech seldom results unless there be profound mental reduction associated. The capacity for restitution of speech faculty in adults by education of the right hemisphere speech centres, however, varies greatly in different individuals.

In the process of silent thought words are revived in the auditory word centre, and there is usually a simultaneous revival in the visual word centre. In some persons the revival in the latter centre comes to be of greater importance than that in the auditory word centre, and people may be classed as "visuals" and "auditives" as regards the revival of words in thought, the majority being "auditives."

Similarly in the processes of spontaneous speech and writing words are revived primarily in the auditory word centre, and the incitations for the revival of the visual and kinæsthetic word memories proceed thence by the commissural fibres. In some people, however (strong visuals), the visual word centre has an importance almost equal to that of the auditory word centre in the initial revival of words for speech. Comprehension is aided by the synchronous revival of word images at several of the speech centres—for an instance, a difficult passage is better understood when read aloud.

In interpreting the clinical aspect from the lesions found after death in cases of aphasia, this variability in the relative importance of the visual and auditory word centres must be borne in mind.

Acute cerebral lesions as a rule produce, by some inhibitory process, a very widespread lowering of function in parts of the cerebrum which are not structurally injured, and until this has passed off correct inferences of the nature and situation of the lesion cannot be made. Cases of aphasia examined for the first time long after the occurrence of the lesion, when compensatory processes are going on, are a frequent source of false deductions, as are also cases in which widely-spread partial lesions in the speech centres occur.

The commonly used terms "sensory aphasia" and "motor aphasia" refer to defects of the afferent and receptive mechanism of speech (auditory and visual word centres and the afferent fibres leading to these), and of the efferent centres and tracts (glosso-kinæsthetic and cheiro-kinæsthetic centres and pyramidal tract) respectively. Word blindness and word deafness are known as sensory aphasia, whereas aphasia (motor vocal aphasia) and agraphia are called motor aphasia. These terms, however, have the disadvantage that they are incorrect, for word deafness, due to an isolated lesion of the auditory centre, usually entails aphasia, and word blindness entails agraphia. Further,

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it is a matter of considerable doubt whether the term "motor" is logically applicable to any of the centres of the cerebral cortex.

The defects of speech produced by lesions of the physiological mechanism that has been above described are classified as follows:—

Lesion of	*Speech Defect Produced.*
1. Motor centres in the bulb and associated neuro-muscular apparatus.	Dysarthria.
2. Pyramidal fibres for speech.	Apnemia.
3. Cheiro-kinæsthetic centre.	Agraphia.
4. Glosso-kinæsthetic centre.	Aphasia. (Motor vocal Aphasia.)
5. Visual word centre.	Word blindness. (Alexia). Sensory Aphasia.
6. Auditory word centre.	Word deafness. Sensory Aphasia.
7. Commissures between centres.	

Amnesia verbalis.

1. Dysarthria

Under this term are included all speech defects which are produced by lesions of the nerve cells of the bulb, their nerve fibres, and the muscles they supply. Dysphonia, dysphagia, and regurgitation of food through the nose from paralysis of the larynx, pharynx, and palate are often associated with this condition, which may present every degree of severity, from the slightest alteration in the pronunciation of consonantal sounds to complete inability to articulate (anarthria). The lesion may be situated in the muscles of articulation, as in the facial type of myopathy; in the peripheral nerves, as in bilateral facial palsy and in diphtherial palsy; or in the cells of the bulbar nuclei, as in bulbar paralysis. In the latter case the whole of articulation is defective; in the first two cases labial articulation, and in the third palatal articulation, is alone in fault. In slight cases of dysarthria a careful analysis of the articulatory defect will reveal which part of the articulatory mechanism is defective, whether tongue, lips, or palate, and it is often useful to make the patient repeat a word several times in rapid succession, when the defect, at first perhaps unnoticed, becomes prominent. The most useful test-sounds for palatal defects are final "b" and "g," which are pronounced "m" and "ng"; the words "rub" and "egg" becoming "rum" and "eng"; for lingual defects the explosives "d" and "t," which lose their explosive character; and for labial defects initial "w" or "wh," as in "went," "which."

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The most severe conditions of dysarthria are met with in the diseases progressive bulbar paralysis and the bulbar form of amyotrophic lateral sclerosis, in tumours of the medulla, and in myasthenia gravis. In the last of these diseases the dysarthria is peculiar in that while it is not present when the patient begins to speak it rapidly appears, and increases the longer the articulatory apparatus is exerted, until almost complete anarthria results. After a short rest articulation again becomes normal. Slighter forms of dysarthria are met with in myopathy, chorea, diphtherial palsy, Friedreich's ataxia, disseminated sclerosis, and general paralysis. In the last three diseases the defect of articulation is often most varied and difficult of analysis, but it is always a combination of the following primary faults:—

1. Slurring; 2. elision of syllables; 3. stumbling over syllables; 4. reduplication of syllables; 5. undue separation of syllables (scanning speech).

Thus some patients with disseminated sclerosis will show many of these defects in saying the words "mutual eligibility," which they pronounce "mewsh-ledg-bil-t-ty." The word groups "West Register Street" and "The Royal Irish Horse Artillery" are very good test phrases for analysing such dysarthric defects.

2. Aphemia

These defects of speech are produced by subcortical lesions damaging the pyramidal fibres which subserve speech in any part of their course from the cerebral cortex to the medulla. They are rarely severe and usually transient, and this is explained by the facts that nerve fibres are more resistant to injury and more capable of recovery from partial damage than is the gray matter, and that when the lesion is unilateral there is another path open to the impulses from glosso-kinæsthetic centre to medulla—that *viâ* the corpus callosum and opposite pyramidal tract. When, however, there is a bilateral lesion of the pyramidal fibres, or where the glosso-kinæsthetic centre is isolated from all its efferent connections—as by a lesion immediately beneath the cortex of the third frontal convolution cutting both the callosal and the pyramidal fibres of the glosso-kinæsthetic centre—the condition may be a permanent one. In the latter case the condition could not be clinically distinguished from that produced by a lesion of the glosso-kinæsthetic centre itself.

The majority of the transient disorders of speech which accompany

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hemiplegia of acute onset are of an aphemic nature. There is no impairment of intelligence and no defect on the receptive side of speech, namely no difficulty in understanding spoken and written words. An aphemic patient knows perfectly what he wants to say, and makes the attempt to express himself with more or less success. He at once attempts to reply to a question, and has no more difficulty with nouns than with other parts of speech. His writing powers are not impaired. The speech centres are themselves healthy, but the way out is more or less blocked.

The speech in partial aphemia resembles that of dysarthria, but there are no signs of local defects in the articulatory mechanism, and hemiplegia on one or both sides is as a rule present.

3 and 4. Aphasia and Agraphia

These terms are confined to defects resulting from lesions of the kinæsthetic speech centres. These have been above described as the centres where thought is clothed for expression, and from whence the motor centres are put into action, the kinæsthetic memories being roused by incitations starting in the higher auditory and visual word centres. Consequently lesions of these centres obliterate the memory of words spoken and written, and the expressive side of speech is lost, while the receptive side of speech—the memories of words heard and seen—is retained in the undamaged auditory and visual word centres. There is usually some degree of mental impairment, for the auditory word centre loses the strong reinforcement produced by glosso-kinæsthetic revivals, and the blocking of the most important path of outflow for its activity produces physiological embarrassment.

In aphasia the loss of the ability to speak may be complete, or some degree of speech may be retained. Such retained speech must be due either to a part of the chief glosso-kinæsthetic centre remaining undamaged or to compensatory activity of that of the opposite cerebral hemisphere. It does not usually amount to more than a few words, which are used irrelevantly and in answer to every question. "Yes," "No," some interjections, and oaths, are frequently retained.

A person suffering with aphasia has little mental impairment. He understands everything that he hears and reads. He may be able to express himself perfectly in writing, though, from the contiguity of the cheiro-kinæsthetic centre and the glosso-kinæsthetic centre, a lesion damaging the latter usually involves the former as well, and

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aphasia is usually, though not necessarily, associated with agraphia. He is conscious of his inability to speak, and recognises that such words as he can use are wrong. Aphasia is frequently associated with word blindness, especially when the lesion is due to a thrombosis of vessels; for the visual word centre is situated at the confines of the blood supply from the middle cerebral artery anteriorly and the posterior cerebral artery posteriorly, and is therefore one of the most likely situations for thrombosis to occur. Such association of aphasia with alexia is always due to the presence of lesions both in the angular gyrus and in Broca's convolution. An isolated lesion of Broca's convolution does not entail alexia.

In the process of recovery from aphasia it is of great interest that those words return first of which the memories are most deeply impressed. For an instance, a person who is able to speak two languages in his recovery from aphasia regains his native tongue long before he regains the later acquired language.

Agraphia occurs almost universally associated with aphasia on account of the very close proximity of the centres above described, and while cases of aphasia without agraphia have been recorded clinically and verified post-mortem, a case of pure agraphia without aphasia has not been verified pathologically. Agraphia entails inability to express thought in writing with either hand, since the kinæsthetic memories for writing are lost, and it is associated also with inability to copy print into writing (transfer writing), though slow copying of letters, just as any outline is copied, is possible. There being no word blindness, expression by means of arranging block letters is possible (typographic writing).

5. Amnesia Verbalis (Sensory Aphasia)

WORD DEAFNESS

Damage to the auditory word centre in certain people (strong auditives), if extensive, is associated with a most profound loss of speech faculty, both on the receptive and on the expressive side. Marked mental impairment is present, since the primary revival of words—the counters of thought—can no longer take place in the usual manner. Though there is no deafness, the memory and meaning of the words heard is lost, and spoken language sounds, to a person suffering from word deafness, like an unknown tongue. The glosso-kinæsthetic centre, deprived of the driving and guiding power, produces nothing more than mere gibberish, and aphasia is

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present. The visual word centre, deprived of its leader, possesses a lowered functional capacity, and there will be difficulty in the perception and expression of written speech.

In other persons (strong visuals), in whom the recall of words in silent thought in the visual word centre is of equal or even of greater importance than recall in the auditory word centre, there is much less interference with speech. For the visual word centre may be sufficient for the recall of words in silent thought, and it may be able to drive the glosso-kinæsthetic centre by an unusual path (in Fig. 14). In a strong "visual" the speech defect may be limited almost to pure word deafness, with little or no aphasia, and neither word blindness, agraphia, nor much mental impairment.

These two conditions produced in different persons, strong auditives and strong visuals respectively, are at the extremes of a series of clinical pictures, passing gradually from the one to the other in accordance with the different relative activity of the auditory and the visual word centre in each person. In all lesions of the auditory word centre, however, the one essential feature is the presence of word deafness. On account of the close proximity of the auditory and visual word centres, and because of the peculiarities of the vascular supply of the angular gyrus above mentioned (p. 145), a lesion injuring the one centre is very prone to affect the other to a greater or less extent. Hence word blindness and word deafness are commonly associated.

In cases where the lesion of the auditory word centre is incomplete, the word deafness may be partial only, and then there is a loss of the words connected with concrete ideas—proper names, and nouns, while the other parts of speech which are more connected with abstract ideas are retained.

A partly damaged auditory word centre, in which the volitional revival of words is impossible, may be roused into activity by stimuli coming from the general auditory centre and the other speech centres. Thus a patient who is word deaf and unable to speak may be able to read aloud perfectly—here the auditory word centre is roused into activity by incitations coming from the visual word centre, and such a patient may be able to repeat words he hears, the stimulus here coming from the general auditory centre. In word deafness there is inability to name objects, and in partial word deafness, while unable to name an object shown him, a patient may express its nature by a circumlocution of words in which he avoids nouns. Thus, when shown a pen, a patient was unable to name it, but said, "It is to write with."

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Word deafness in any but its slightest forms renders the patient unaware of his errors, and in this respect strongly contrasts with aphemia and aphasia, for in the latter conditions errors are recognised by the patient. Partial lesions of the auditory word centre frequently lead to an irregular and inco-ordinate revival of words. Wrong words may be used—a patient may say "dog" when he means "cat," and is unaware of his error. In other cases words are wrongly arranged, so that they carry no meaning, and in the most severe cases in which this irregular revival occurs speech may be reduced to a mere jargon. These phenomena are termed "paraphasia." Word deafness rarely remains permanent in severe degree, as partial recovery tends to take place in the damaged centre and compensation occurs by the raising into activity of the right auditory word centre.

Lesions in both right and left auditory centres produce more or less complete deafness for sounds, with complete and lasting word deafness, aphasia and profound mental impairment.

WORD BLINDNESS (ALEXIA)

Lesions of the left visual word centre produce inability to recognise the meaning of written words, which then appear as hieroglyphics, since the visual memories of words are lost. There is also agraphia present, for, as has been above shown, the cheiro-kinæsthetic centre is driven by the visual word centre. In an isolated lesion of the visual word centre speech is but little interfered with; there is little or no mental impairment, and both the receptive and the expressive mechanism for spoken speech are uninterfered with. Except in the rare cases of very strong "visuals" there is no difficulty in naming objects, and no paraphasia. Where the damage to the visual word centre is partial the alexia and agraphia may be partial, and in writing there may be an irregular revival of words, so that wrong words are written, or words are written in a wrong order, this defect constituting "paragraphia." A person suffering from alexia may be unable to recognise a single letter (letter blindness), or, while able to recognise letters, he may be unable to recognise words. Often the power of recognising numerals remains, and a patient may be able to add, subtract, and multiply simple numbers correctly while he is unable to recognise letters. The power of recognising his own name is sometimes preserved by a patient who is unable to read any other word.

A destructive lesion of the visual word centre necessarily entails

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inability to read aloud and inability to write from dictation or to copy print into writing (transfer copying).

From the proximity of the angular gyrus to the half vision centre in the occipital lobe and to the posterior limb of the internal capsule, word blindness is frequently associated with right hemianopsia.

There remains to be described under this heading cases of word blindness which are unassociated with agraphia (pure word blindness). Such patients are able to express their thoughts perfectly in writing, while they are unable to read what they have written. In these cases there is no damage to the visual word centre, but the lesion is in such a position as to cut off all the afferent connections of the visual word centre, namely the paths connecting it with both half vision centres and with the angular gyrus of the right cerebral

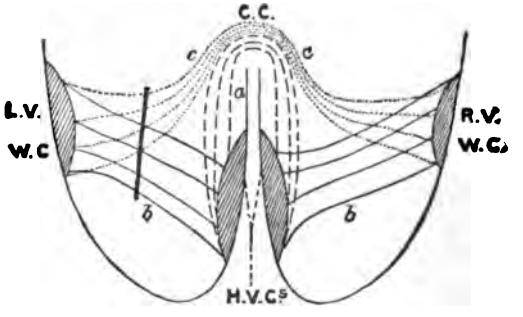

FIG. 15.—A diagram representing the mode of production of pure word blindness. *C.C.*, posterior extremity of corpus callosum; *c.c.*, commissural fibres connecting the two visual word centres; *b.b.*, fibres connecting each half-vision centre with the visual word centre of the same side (Bastian).

hemisphere, while the connections with the auditory word centre and the cheiro-kinæsthetic centre are intact. The lesion producing pure word blindness is a subcortical lesion extending deeply behind the angular gyrus, and, as such a lesion must necessarily implicate the optic radiation, pure word blindness is always associated with hemianopsia, or, when partial, with hemiachromatopsia (see Fig. 15).

It has been above pointed out that from the proximity of the auditory and visual word centres, lesions in the neighbourhood of the posterior end of the Sylvian fissure are apt to damage both centres. When this occurs word deafness, word blindness, aphasia, agraphia, and much mental impairment result, and the understanding of gestures, and expression by means of gestures, alone are left. In these cases, however, some degree of speech may be ultimately acquired by the education of the speech centres in the right hemi-

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sphere. The compensatory capacity of the right hemisphere speech centres presents great individual variation.

LESIONS OF THE COMMISSURES BETWEEN THE CENTRES

From a consideration of the relative inter-dependence of the speech centres it is obvious that damage to the commissures between the centres will produce speech defects. These are often of a peculiar nature. A few of these only can be briefly mentioned.

Of the two commissures connecting the auditory and visual word centres damage limited to the audito-visual commissure will produce inability to write from dictation, with slight agraphia, without other speech defects, while an isolated lesion of the visuo-auditory commissure produces inability to read aloud, speech being otherwise perfect.

When both these commissures are damaged there is inability to read aloud, inability to write spontaneously or from dictation, while the power of transfer copying is retained.

OBJECT BLINDNESS AND AMIMIA

The most severe degrees of speech defects are sometimes accompanied by a loss of the primitive forms of cognition and expression. The term "object blindness" is used to denote inability to recognise the nature and use of objects, while "amimia" denotes inability to express thought by signs and gestures. These signs are generally associated with severe mental impairment, and are present where extensive lesions of the chief speech centres in both hemispheres have occurred. As transient signs they are frequently present after the occurrence of lesions damaging both the auditory and visual word centres on the left side.

Etiology — *Organic lesions.* — The most frequent cause of speech disorders is occlusion of the left middle cerebral artery or some of its cortical branches by thrombosis or, less commonly, by embolism, which, on account of the almost complete absence of anastomosis between the arteries of the cortex, leads to more or less complete evascularisation and subsequent softening of the area supplied by the affected vessel. The middle cerebral artery at the commencement of the posterior limb of the fissure of Sylvius divides into five branches, the first of which passes forwards to supply the frontal convolution at its posterior part—an isolated lesion of this vessel produces pure aphasia. The second and third branches

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supply the central convolutions (motor area), while the fourth branch passes backwards along the posterior limb of the Sylvian fissure and supplies the upper temporal convolution in its hinder part and the angular and supra-marginal convolutions. Occlusion of the fourth branch alone produces word blindness and word deafness; but the latter is usually not complete, for the hinder part of the posterior temporal convolution also receives blood from the fifth branch. Occlusion of the whole left middle cerebral artery in a right-handed person will produce complete loss of speech, both receptive and expressive, with severe right hemiplegia. Other important causes of speech disorders are the occurrence of cortical hæmorrhage, meningeal hæmorrhage, direct injury from blows on the head, abscess, new growths, encephalitis and meningitis in the neighbourhood of the chief speech centres.

Functional defects.—In the absence of any recognisable structural change in the speech centres, defective function must be referred to nutritional and metabolic disorders. Such an affection of both glosso-kinæsthetic centres produces hysterical mutism. Speech disorders of this nature may follow emotional shocks and occur sometimes in poisoning by belladonna, stramonium, and snake-venom. The transient aphasia following attacks of epilepsy and Jacksonian epilepsy must be placed in this group. Functional defects of speech as a rule are attributable to disorder of the glosso-kinæsthetic centre only. These are mostly transient and are not infrequently recurrent.

Prognosis.—In attempting to estimate the extent to which recovery may take place it is necessary first to bear in mind that sudden cerebral injury is apt at first, by some process which may be called "inhibition," to cause loss of function in centres which are not structurally damaged. Such inhibitory phenomena last usually not longer than a week, and until they have passed off it is impossible to make a definite statement as to recovery. Speech may be regained by two entirely separate processes—either by recovery of function in a partly damaged centre, or by compensatory activity in the secondary speech centres of the undamaged hemisphere.

The possible recovery of function in a centre will depend upon the nature of the lesion and its extent. It will be greater where a lesion may be judged to be one of pressure rather than of actual destruction, especially if such pressure be removable, as in meningeal and sub-cortical hæmorrhage, abscess, and gumma, and least when widely-spread arterial disease and a failing heart suggest that

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the lesion is thrombotic. The greater the extent of the lesion, if it be presumably from vascular occlusion, as judged by the associated symptoms, paralysis, anæsthesia, and hemianopsia, the less is the chance for functional restitution, as there is then little hope of the restoration of circulation through collateral vessels. Where the lesion is one involving the fibres connecting the speech centres, as, for an instance, a hæmorrhage into the left internal capsule, producing aphemia, the probability of recovery is much greater than when the speech centres are damaged, as the tracts are more resistant to injury than are the centres, and there is always the possibility of the opening up of fresh associational paths.

The capacity of transference of function from a damaged speech centre to the corresponding centre in the other hemisphere by a process of re-education varies so much in individual cases that very few definite statements can be made with regard to prognosis, but as a rule the younger the patient the more certainly does this compensation occur. Thus in children under the age of six years unilateral lesions produce no permanent speech defect, but even to this rule some important exceptions have been met with. On the other hand, total destruction of the left auditory and visual word centres in adults has, in a few instances, been followed by an almost complete restoration of speech. Some idea of the possible degree of restoration of speech by compensation may be gained by the examination of the patient when this process is taking place, from the nature of the restoration, and from its rate of progress.

Treatment of speech defects.—A careful and patient system of re-education in speech, such as is used in the teaching of mentally deficient children, is often of great value in all forms of speech defect. From the amount of labour that the teacher has to expend for very little apparent progress made, this treatment is not often given a fair trial. A fair degree of mental intelligence must be present, and care must be taken that the lessons are not prolonged to the production of the mental fatigue and confusion which occurs so readily in aphasic patients. The patient should first be taught to utter a simple vowel sound, then the several vowel sounds, and then consonants and their combinations, and should be directed while learning to imitate the movements of the lips and tongue of the teacher. An intelligent patient very soon comes to recognise that he has no paralysis of the articulatory mechanism, but has simply lost his memory of words.

Testamentary capacity.—No rule can be laid down as to the capacity of a person suffering with speech defects to exercise civil

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rights and to make a will, but each case must be judged separately. The first consideration is the degree of intelligence, and when this is good it is essential for such capacity that there is some mode of cognition and of expression left. Pure word blindness, and the extremely rare condition pure word deafness, do not interfere with the exercise of civil rights, for the patient can understand what he hears in the first case, and what he reads in the second case, and in both cases can express himself in speech and writing. In cases of pure aphasia and pure agraphia there is complete civil capacity, but when these are present in the same case, though intelligence and the receptive side of speech may be little impaired, the expressive side of speech is reduced to gesture, and the most extreme difficulty may be met with in interpreting the patient's wishes.

Auditory amnesia and co-existing auditory and visual amnesia, except in the slightest forms, interfere with testamentary capacity and capacity for exercising civil rights. There is here great loss on both receptive and expressive sides of speech, loss of memory, and considerable mental impairment.

The **method of examination** of patients suffering with speech defects should be in accordance with some definite scheme so drawn up as to test each part of the complex physiological process of speech.

The following scheme is abbreviated from that drawn up by Dr. Bastian :—

1. Is the person right- or left-handed, and, if the latter, does he write with the right hand?
2. What is the state of education as regards reading and writing?
3. Does he understand the nature and uses of objects, and can he understand or express his wants by pantomime and gesture?

The Activity of the Auditory Word Centre and the Glosso-Kinæsthetic Centre and their Connections

4. Is he deaf? If so, to what extent, and on one or both sides?
5. Can he recognise ordinary sounds and noises?
6. Can he comprehend speech? If so, does he at once attempt to answer a question?
7. Is spontaneous speech good? If not, to what extent is it impaired? Does he make use of recurring utterances, wrong words, or gibberish?
8. Can he repeat words uttered before him?

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The Activity of the Visual Word Centre and the Cheiro-Kinæsthetic Centre and their Connections

9. Is the sight good or bad? Is there hemianopsia or optic neuritis?
10. Does he recognise printed or written words? If not, does he recognise single letters or numerals?
11. Can he write spontaneously? What mistakes occur in writing? Is there paragraphia? Can he read his own writing some time after it is written?
12. Can he copy written words, or from print into writing? Can he write numerals, or perform simple mathematical calculations?

The Associated Activity of the Speech Centres

13. Can he read aloud?
14. Can he name at sight words, letters, numerals, and common objects?
15. Can he write from dictation?

Impediments of Speech

1. Stammering or stuttering.—"A spasmodic affection of the muscles concerned in articulation, occasioned by erroneous nervous control, leading to a sudden check in the utterance of words, or to a rapid repetition of the consonantal sound in connection with which the difficulty arises" (Colman).[1]

The causes of this disorder are very obscure. It is much more common in boys than in girls, and a neurotic tendency is frequently to be observed both in the family and in the stutterer himself, apart from the speech defect. The disorder is rarely congenital, and may come on at any time between early childhood and puberty. In some cases it has appeared after severe mental emotions, and in others after acute diseases, especially measles and diphtheria. Imitation is an occasional cause.

The condition is not associated with any structural change in the nervous system, nor in the organs of articulation.

In articulate speech three muscular mechanisms are concerned: (1) the respiratory mechanism for supplying the blast of air; (2) the larynx for producing the voice, and (3) the muscles of the lips, tongue, and palate for articulation. For distinct speech there must be absolute co-ordination of these mechanisms one with another.

[1] "Impediments of Speech," Allbutt's *System of Medicine*, vol. vii.

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Stuttering is essentially due to defective accord between the vocal and articulatory mechanisms, the laryngeal mechanism lagging behind the oral in the production of certain consonants. Consonants are, in nearly all cases, the source of difficulty in stuttering, and while they are all buccal sounds, some begin with a laryngeal sound, while others are purely buccal. These are termed "voiced consonants," B, W, V, Sh, Z, Th, D, L, R, G, Y, and voiceless consonants, P, F, Th, S, Sh, T, K, respectively, while N, M and Ng are "voiced nasal resonants." If one articulates these consonants, it becomes at once clear that it is the presence of the laryngeal element or "voicing" which makes the difference between B, V, Z, D, G, and P, F, S, T, K.

A careful attention to the manner in which the letter sounds are produced is absolutely essential in the investigation and treatment of stuttering. The difficulty occurs most commonly with the explosive consonants P, B, T, D, G, K, and nearly always where these occur as initial letters; that is, in starting the articulatory mechanism, and to avoid this difficulty arising after a pause, most stutterers speak in a rapid, monotonous fashion. The difficulty in the most part lies in the direction of energy to articulation rather than to phonation. The patient most often remains silent during his attempts to speak, but occasionally he produces the first sound and continues to repeat it. Often the patient uses tricks to prevent the stutter, and these become grafted upon him as (1) associated sounds—whooping, grunting, crowing, etc.; (2) habit spasms—contortions of face, limbs, and body. The trouble occurs almost entirely in consonantal speech, and not in singing or in intoning.

Prognosis.—Most cases tend to a spontaneous cure, and recovery is hastened in all cases by systematic treatment. The worst subjects are those of a nervous and sensitive temperament.

Treatment.—The patient should be removed as far as possible from causes increasing his self-consciousness of his defect, and should have daily reading exercises from an intelligent teacher.

The following system for such exercises is given by Dr. Colman[1]:—

(1) The chest must be kept well filled with air. This most important point is often most difficult to the patient.

(2) The patient must speak slowly, with a full resonant voice.

(3) When he comes to a word on which he tends to stutter, he should raise his voice and direct his energies to vocalisation and not to

[1] "Impediments of Speech," Allbutt's *System of Medicine*, vol. vii.

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articulation. If the difficulty be a voiced consonant, he must be directed to "voice" it. If the consonant is voiceless, his attention must be directed to the vocalisation of the subsequent vowel sound. For instance, in "pat" he must attempt to vocalise the "at," and he will find little difficulty in prefixing "p" as the syllable is uttered.

(4) Gymnastic and singing exercises are valuable additions to treatment, and should there be associated movements present, the reading exercises should be carried on in front of a mirror, so that the patient may be aware of these himself and endeavour to suppress them.

The results of treatment often come very slowly at first, but perseverance in nearly all cases will spell success.

2. LALLING.—A defect due to want of precision in the action of the oral articulatory mechanism.

This condition characterises the speech of children before articulation is completely learnt. As a condition persistent from infancy it occurs chiefly in connection with defective intelligence.

3. LISPING.—A defect due to the indistinct enunciation of certain consonants, or to the substitution of wrong consonants.

This condition, which occurs normally in infants learning to speak, is due either to clumsiness of articulation, which has become a habit, or to defective conformation of the mouth.

4. IDIOGLOSSIA.—A condition in which, from difficulty in learning the pronunciation of certain consonants, a child uses other consonants, and so seems to speak a language entirely his own.

The following admirable illustration is given by Dr. Colman from one of his patients who thus repeated the Lord's Prayer.

"Ouë Tahde na ah in edde, anno de Di na, I tidde tah, I du de di on eet a te e edde, te ut te da ouë dade ded, e didde ouë tetedde a ne ahdin to te tetedde adase ut, ne no te tetate, ninne utte enu, to I ah te ninne, pouë e dordy, to edde e edde. Amé."

This patient was unable to pronounce many of the consonants, and substituted for them "t," "d," or "n."

The treatment of the three defects, lalling, lisping, and idioglossia, consists in patient and careful teaching of pronunciation. The prognosis in idioglossia is invariably good.

5. APHTHONGIA.—A condition in which the attempt to speak sets up severe spasms in the muscles of articulation, chiefly of the tongue. Nothing is known as to the pathology of this rare disease, which seems to be of the same class as writer's cramp, violinist's cramp, etc.

J. S. COLLIER.

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DISEASES OF THE SPINAL CORD AND ITS MEMBRANES

It has been already pointed out (p. 34) that as no actual division is anatomically possible between the spinal cord and the brain, so no real distinction can be made between cerebral and spinal disease; convenience in treating the subject alone justifies any attempt at separation.

Sufficient has been said as to the general causation of affections of the central nervous system (p. 42), and it is only needful here to indicate that in the investigation of spinal cord diseases many etiological factors must be kept in view as possibilities—to wit, vertebral disease due to caries, old injury, or it may be cancer; syphilis, the commonest cause perhaps, and the most varied in its mode of action; tubercle; new growths; pre-existing infectious diseases; constitutional conditions, such as anæmia, diabetes, and chronic intoxications; nervous heredity, whether of the particular disease or of a more general kind; exposure and exhaustion, physical or mental. The outbreak of chronic progressive diseases, like disseminated sclerosis or paralysis agitans, is often associated by patients with mental and moral stress, such as terror, grief, anxieties, etc. Of many spinal diseases the cause is as yet utterly unknown; and further it may be added, by way of caution, that symptoms due to hysteria may so closely resemble those due to organic spinal lesion as to render the diagnosis difficult, sometimes almost impossible.

General Morbid Anatomy

For the immediate results of injury to the cord reference may be made to works on surgery. It suffices to say that a purely local disorganisation due to traumatism may extend its influence, either within a few days by means of a local myelitis spreading by contiguity from the seat of injury, or after a lapse of weeks or months by means of a secondary degeneration of the ascending and descending tracts.

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Nor need the lesions of bone produced by cancer or caries of the vertebræ be described. Both these affections tend to spread inwards: cancer with the greater rapidity and certainty, involving and destroying the nerve roots, membranes, and cord itself; caries with less formidable strides. The cord suffers, in caries of the vertebræ, in the following way:—At the level of the vertebral lesion a chronic inflammation of the meninges is set up, and these, commencing with the dura, become thickened and fused together into an annular mass. The cord is pressed upon by this ring, or sometimes by pockets of pus and debris which form in connection with the caries, or it may even be by vertebræ which have been actually dislocated. It thus becomes thinned, and eventually either invaded by the inflammatory process going on around it, or softened owing to blockage of its nutrient vessels in the meninges. The process, therefore, has been aptly called a "compression-myelitis."

MENINGEAL HÆMORRHAGE, either inside or outside the dura, a possible result of injury, of blood states, such as purpura, or of very acute meningitis, is a rare condition which need only be mentioned.

ACUTE MENINGITIS, when local, may also be caused by injury or by some local disease, such as necrosis of a vertebra. Acute general meningitis is commonly a manifestation of septicæmia or of some specific disease. Generally, the membranes of the brain are affected as well. Its seat is the internal membranes, viz. the pia and arachnoid. The earliest stage, not often seen, nor, indeed, easily recognisable, doubtless consists in a hyperæmia of the membranes and surface of the cord. From this we must distinguish the mere fulness of spinal veins found in most post-mortems where the body has lain on the back, and due simply to gravitation. The next stage is characterised by exudation. Fluid of a dirty or even sanious appearance may run out when the dura is slit up. But that which is really distinctive is an exudation into the meshes of the arachnoid, which covers in the cord and adheres to its surface even when removed from the body. It may be purulent or semi-solid, and is most abundant upon the posterior surface of the cord, and in the lower dorsal and lumbar regions. Indeed there may be none in the cervical region, so that a hasty inspection of this part is not enough to exclude spinal meningitis. The exudation should be examined for micro-organisms. The cord itself in this stage of meningitis often looks pale and softened rather than hyperæmic.

SUBACUTE SPINAL MENINGITIS is mostly either tuberculous or syphilitic. *Tuberculous meningitis* is the more acute, and anatomically it resembles the same disease in the cerebral membranes,

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which, indeed, it accompanies, forming a subsidiary factor thereof. In some rare cases, however, the spinal lesion predominates, both clinically and anatomically.

Syphilitic meningitis, to judge from clinical data, is frequent, but post-mortem observations upon it are not very common. It is not generalised, but usually consists in a localised patch or patches of thickening, involving the pia-arachnoid, due to infiltration with small cells (gummatous deposit). The connective-tissue sheaths that dip into the cord are also infiltrated; the vessels may be affected with a gummatous endarteritis. Thus, whether from spread of the inflammation inwards (meningomyelitis), or from blocking of its nutrient arteries, a segment of the cord becomes softened, and there results a common form of "transverse myelitis." Other manifestations of the syphilitic process may occur. Thus a mass of gummatous tissue, sufficiently large to be called a tumour, may spring from the membranes and compress the cord, or gummata may form on the nerve roots, or on the internal aspect of the dura. These processes when obsolete are represented by thickenings, scarrings, or adhesions of the parts which have been attacked.

In CHRONIC MENINGITIS there is thickening of the pia, and possibly adhesions between the internal membranes and the dura, rendering it difficult to peel the latter off; the process may be local, in which case a syphilitic origin is probable, or more diffuse. Perhaps chronic alcoholism may cause a thickening of the spinal membranes, as it undoubtedly does of the cerebral. In cases of old-standing spinal degeneration the pia may become thickened over the site of the diseased tracts. Such a chronic meningitis of the posterior aspect of the cord may sometimes be seen in tabes.

Mention may here be made of the rare disease known as PACHYMENINGITIS CERVICALIS HYPERTROPHICA. This affects the membranes chiefly at the level of the cervical enlargement. Layers of connective tissue form upon the inner surface of the dura, and increase in thickness, till a veritable tumour of annular shape encircles, compresses, and may even partially involve the cord. The nerve roots are involved, the arachnoid and pia become fused with the mass of new tissue, and the cord itself in some cases is disorganised. The origin of the disease is not yet definitely known; some consider that it is only a special localisation of disease (such as syphilis), which may involve other regions of the cord.

Diseases of the cord itself may be roughly classified into the acute and the chronic. This division corresponds, with some ex-

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ceptions, with the division into diseases that are inflammatory or necrotic on the one hand and those that are degenerative on the other. Again, they may be classified according to their distribution, whether this be local, diffuse, disseminated, or selective (systemic).

ACUTE DISEASE OF THE CORD, "ACUTE MYELITIS," may be due either to inflammation or to necrotic disorganisation, which has been brought about by obstruction of the vessels. The result in either case is softening, and it may be impossible, unless the case be very recent, to say which cause has produced the softening. The cord, when softened from acute myelitis, loses its consistency, becoming less firm to the touch or even diffluent. On section the cut ends bulge, the distinction between grey and white matter is blurred or obliterated, and the colour is creamy white, unless there has been recent acute inflammation, when it may be reddened or even hæmorrhagic. The preparation of proper microscopical sections may be frustrated by difficulties in hardening the specimen. The main microscopical changes appear to be that the myeline sheaths break up and separate into small particles or larger balls of fat (staining black with Marchi's fluid); that the axis cylinders become swollen and may finally disintegrate; that the ganglion cells may swell, become structureless or vacuolated, and eventually shrivel up. Soon large cells become visible, full of granular fat and detritus; and perhaps also large amorphous deeply-staining cells known as corpora amylacea. In very severe myelitis the neuroglia and connective tissue may also break down. When the process is one of recent inflammation, the blood vessels are engorged, the tissues are infiltrated with leucocytes, perhaps even with red blood corpuscles, and Deiters' cells are increased in number.

Softening limited to one level of the cord, but involving indiscriminately all the elements at that level, is called "*transverse myelitis*." It has been explained how this may result from disease, cancerous, carious, or syphilitic, of the adjacent vertebræ or meninges. Doubtless it may be also caused by arterial thrombosis, though this is difficult to demonstrate. It is most common in the dorsal region. "*Acute general myelitis*" is far rarer. In this the process starts usually from the lower part of the cord and spreads rapidly upwards, involving all the structures as it proceeds. Recent observations appear to indicate that it is due to some bacterial infection. Still rarer is the form known as *acute disseminated myelitis*, wherein small foci of disease are found scattered indiscriminately through the cord, or through the nerve centres generally.

There is one instance in which acute myelitis appears to be

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selective, *i.e.* to limit itself to certain special constituents of the cord. This is *acute anterior poliomyelitis*, known clinically as infantile spinal paralysis, the result of which is destruction of the large multipolar cells of the anterior horns. It is still somewhat uncertain how this result is brought about. According to one view it begins as an acute degeneration of these cells, and when they have perished a secondary shrinking of the tissues around them takes place. According to another view, based on the examination of some rare instances when death has occurred early in the disease, the process is truly inflammatory, and involves not only the cells,

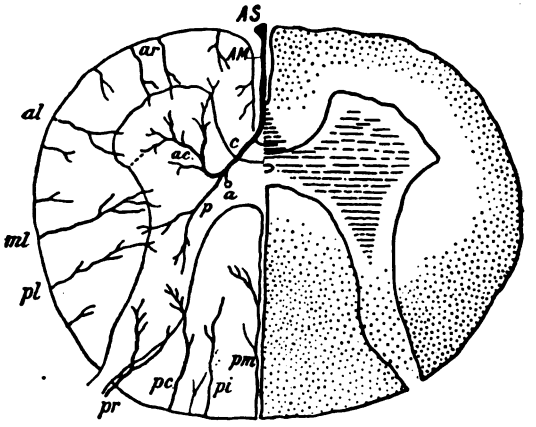

FIG. 16 is a schematic representation of a transection of the spinal cord showing the arterial distribution. (Modified from Williamson.) On the left side are represented the large branch of the anterior spinal artery (*AS*), which supplies the anterior horns of grey matter, and the small arterial twigs from the pia-arachnoid membrane, which supply the peripheral white matter. Some enter directly from the membrane (*al*, *ml*, *pl*), some by the anterior nerve roots (*ar*), and others by posterior root (*pr*). On the right side the dotted area represents the distribution of the peripheral arterioles, the lined area that of the anterior spinal artery, the unshaded being supplied from both sources.

but the whole of the anterior cornu at one level, perhaps even the adjacent parts of the cord. The limitation to the anterior cornu is on this hypothesis largely due to the distribution of the small arteries (terminal branches of the anterior spinal) which converge upon this point. In the later stages, at any rate, the anterior cornu is found to be shrunken and sclerosed, the cells have disappeared, and the anterior nerve roots, the motor fibres of the nerve trunks and the corresponding muscles, which depend for their nutrition upon the cells, are degenerated.

Myelitis may be subacute in its onset, neither chronic nor

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acute in type, but transitional. Thus acute poliomyelitis, especially in adults, may take time to develop, and so may a transverse myelitis (even in cases which we do not suppose to be syphilitic). But of the morbid anatomy we have here little to say, except that the less acute the process, the less likely is softening to be found, and that microscopically multiplication of nuclei and increase of neuroglia will be more prominent features than breaking up of the myeline.

Just as acute spinal disease may result either from genuine inflammation or from other causes, so in chronic spinal disease it is difficult to distinguish anatomically between CHRONIC INFLAMMATION on the one hand and nervous degeneration on the other. Both processes result in the condition called "sclerosis," characterised by overgrowth of the connective tissues and atrophy of the nerve fibres; but either of these last-mentioned factors may be secondary to the other one, and we are often unable to tell, at any rate from mere histology, which came first; though we may gain some information from the distribution of the lesion. For when the disease is selective (systemic), *i.e.* follows the lines of known neurones, the presumption is that it consists not in an inflammation, which must spread either by contiguity or by vascular and lymphatic routes, but in a nervous degeneration. The best example of this is the so-called "secondary degeneration." We know that the nutrition of a nerve fibre depends upon the nerve cell, of which indeed it is but a prolongation, the whole forming a single "neurone," and that this destruction of the cell or permanent severance of it from the fibre causes atrophy of the fibre. Thus, when by disease or injury the cord is cut across, the distal parts of the severed neurones, *i.e.* such tracts of fibres as are separated from their nutrient cells, begin to degenerate, so that below the lesion the long fibres of the descending (pyramidal) tracts, and above it those of the ascending tracts (columns of Goll and direct cerebellar tracts) gradually perish in their whole length. The various stages of this process can be demonstrated by the microscope and suitable stains. The first stage is principally evidenced by disintegration of the myeline sheaths. These, when normal, stain brown under the combined action of bichromates and osmic acid, but when degenerating they break up into discontinuous masses and granules, which stain black like fat (Marchi's method), so that a degenerating tract is marked by dots and balls of black. Next the myeline becomes absorbed; its absence is best shown by stains which bring into relief the normal myeline. Thus Weigert's hæmatoxylin method, which colours a normal tract of fibres dark

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blue, leaves a degenerated tract unstained. Doubtless the axis cylinders perish too, but their fate is more difficult to demonstrate. Lastly, upon the absorption of the myeline there follows an overgrowth of the neuroglia and connective tissues; carmine, which stains these tissues, will now mark the degenerated parts red. This later stage of degeneration can be recognised naked-eye. The affected parts look gray instead of white, and may be firm or even shrunken; and it may in all strictness be called "sclerosis."

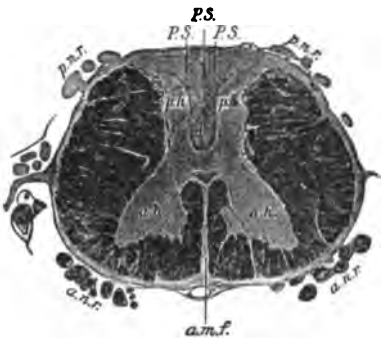

FIG. 17[1] shows the characteristic sclerosis of the posterior columns in *tabes dorsalis*. There is also seen atrophy of the posterior nerve roots (*p.n.r.*) The remainder of the spinal cord is normal. *a.m.f.*, anterior median fissure; *P.S.*, posterior septum; *p.c.*, posterior columns; *p.n.r.*, posterior nerve roots; *a.n.r.*, anterior nerve roots; *a.h.*, anterior horns; *p.h.*, posterior horns; *c.p.t.*, crossed pyramidal tract; *d.p.t.*, direct pyramidal tract.

Such secondary degeneration evidently begins in the nervous parenchyma; this follows both from the distribution and the known causation of the disease. But there are other forms, which similarly spread along definite neurones, and are therefore presumably parenchymatous diseases, though are not secondary to any coarse lesion. These are called "primary degenerations," and are exemplified by some of the most interesting diseases of the cord. In *tabes dorsalis* the posterior nerve roots, and all the system of intraspinal fibres which originates from the nerve cells contained in them, that is to say, the first afferent system of neurones, degenerate. Since these run mainly in the pos-

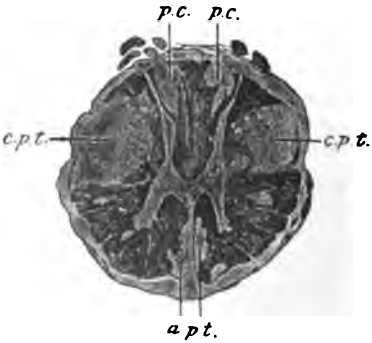

FIG. 18 represents a section of a spinal cord from a case of *combined postero-lateral sclerosis*. Patches of degenerated nerve fibres are seen in the posterior columns (*p.c.*), the crossed pyramidal tracts (*c.p.t.*), and the direct pyramidal tracts (*d.p.t.*). The anterior horns (*a.h.*) are also shrunken.

[1] Figures 17, 18, 20, 21, and 22 are drawn from original preparations in the possession of Dr. W. A. Turner, stained by the method of Weigert-Pal. By this method normal medullated nerve fibres are stained a deep blue, represented *black* in the figures; degenerated nerve fibres (sclerosis) and gray matter are unstained.

terior columns, the disease has been called "posterior sclerosis." By *lateral sclerosis* is meant a degeneration of the efferent neurones as they descend from the cerebral cortex to the anterior cornua of the cord in the pyramidal tracts. A primary, uncomplicated degeneration of the pyramidal tracts is probably very rare indeed, but it occurs in conjunction with disease of the afferent neurones in the posterior columns ("postero-lateral sclerosis," "combined system-degeneration") in *general paralysis*, in *Friedreich's disease*, and in what is known clinically as *ataxic paraplegia*. All these primary degenerations of neuronic systems, "system diseases" as they are sometimes called, may indeed be due to some abnormal condition of the nerve cells at the head of degenerating tracts of fibres; this seems probable, but is not yet proven anatomically, and we are uncertain in what part of the neurone cell or fibre the primary fault lay. There exists, however, a class of diseases (*muscular atrophies of spinal origin*), whereof the most marked anatomical feature is degeneration of the nerve cells in the anterior cornua. Sometimes this is the only spinal lesion present sometimes, *e.g.*, in the variety termed *amyotrophic lateral sclerosis*, the pyramidal tracts degenerate also. This particular affection constitutes indeed the very best example of "system-degeneration," for in it the whole efferent nervous apparatus has been found

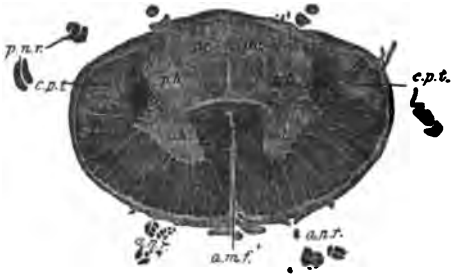

FIG. 19 illustrates the sclerosed areas found in *Friedreich's ataxy* (after Schultze), viz. in the posterior columns (*p.c.*) and the crossed pyramidal tracts (*c.p.t.*).

FIG. 20 is from a case of *amyotrophic lateral sclerosis*. There is sclerosis of the crossed pyramidal tracts (*c.p.t.*), and atrophy of the ganglion cells in the anterior horns (*a.h.*).

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diseased: both the lower neurone, *i.e.* the anterior cornual cells and (by consequence) the motor nerve roots, nerve trunks and muscles which depend upon them for nutrition; and the upper neurone as well, *i.e.* the pyramidal tracts from their lower terminations up through the medulla, internal capsule, corona radiata, etc., to the cortex cerebri, and sometimes even the large cells of the cortex from which these tracts spring.

Early degeneration of nerve cells is best shown by Nissl's method of staining with methylene blue. A normal cell exhibits certain sharply-stained streaks and dots definitely arranged throughout the body of the cell. In a cell that is degenerating, from whatever cause, these bodies at first stain less distinctly and more diffusely than normal, next they refuse to stain, so that the cell body looks more homogeneous and less stained than it should be. The cell also becomes more rounded in shape; the nucleus moves towards the periphery of the cell and may be eventually extruded. Later, the cell may become vacuolated or pigmented, and may at last shrink and disappear. Associated with these structural alterations are certain chemical changes in the tissues which appear, under some circumstances, to give rise to bodies of a severely toxic character, such as cholin and its derivative neurin. Drs. Mott and Halliburton have found the former in the cerebro-spinal fluid of general paralytics, and the possibility of auto-intoxication from such a cause becomes apparent (see pp. 34 to 41).

But chronic spinal disease is not always distributed in the way just described. Instead of mapping out definite systems of neurones, it may involve them indiscriminately. Thus we may have, it would appear, a chronic transverse myelitis limited to a segment of the cord, but affecting all the tissues at that level; or a more diffuse process extending for some length of the cord, and most probably spreading from the neighbourhood of the vessels and connective tissue septa. Since it lacks a definite relation to the nerve elements, we may perhaps term it a chronic interstitial inflammation. A better known and more remarkable type of chronic spinal disease is that known as "*insular*" or "*disseminated*" *sclerosis*. This is characterised by patches of disease varying in size and shape, and distributed quite irregularly through the cord, the brain, and sometimes even in the nerve trunks. These patches are either firm and gray or occasionally less dense and of a lighter colour. Microscopically they exhibit an overgrowth of interstitial tissue, the interstices of which either contain breaking down nerve tissue or merely gaps, from which nerve tissue has disappeared. The centres, pre-

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sumably the oldest portions, of the patches may show little more than a close web of fibrillar tissue. Sometimes a vessel has been noticed to pierce the centre of the patch. In the degeneration of nerve fibres that accompanies disseminated sclerosis, Charcot noticed this peculiarity, that while the myeline disappears early the axis cylinders may persist even in an advanced stage of the process. With this he connected another remarkable fact, namely, that secondary sclerosis of the ascending and descending tracts is not common, even when these tracts have been cut across by a primary patch of the disease. Whether in disseminated sclerosis there is first degeneration of the nervous parenchyma, and then a complementary overgrowth of the neuroglia, or whether the original process be an interstitial inflammation of toxic origin, in the course of which the nerve elements are destroyed, is still an open question.

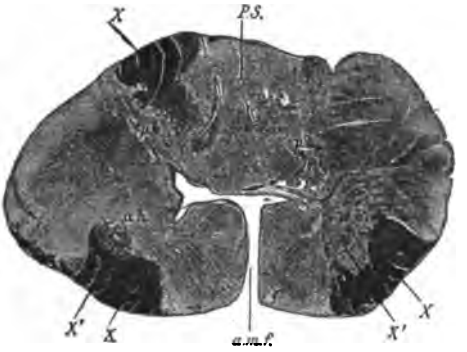

FIG. 21 is taken from an old-standing case of *disseminated sclerosis*. There is sclerosis throughout the cross section of the spinal cord, with the exception of the darkly-stained areas *X* and *X'*.

CONGENITAL ABNORMALITIES of the cord are not common. Heterotopia of the gray matter, *i.e.*, a condition in which the central columns of gray matter are found to occupy an abnormal position, is a pathological curiosity. Similarly, dilatations of the central canal may be found post-mortem, which have given rise to no symptoms. Nevertheless, such enlargement of the central canal, *hydromyelia*, as it is termed, may have some interesting relations. In the first place, it may be associated with spina bifida. Here the neural arches at the lower end of the cord have failed, in the course of development, to meet, and a gap is therefore left. The gap may be only in the posterior aspect of the cord, the bony arches being complete. This condition, "spina bifida occulta," may be unrecognised during life, or revealed only by obscure paralytic symptoms. Or the gap may be in the bones as well, "spina bifida aperta," and then a fluctuating tumour, formed by the incomplete central canal full of cerebrospinal fluid, presents beneath the skin at the

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lower part of the spinal column. In the second place, abnormalities of the spinal canal must be considered in relation to the disease known as *syringomyelia*. This term has strayed a little from its original meaning. Applied at first to all instances of abnormal canals within the spinal cord, it is now practically limited to a condition in which such excavations are but a later development. In this disease the neuroglia, which, in the normal cord, exists in some quantity behind the central canal, proliferates, till it forms what might be called either a central tumour or a central myelitis. The process is most marked in the cervical region, where it may cause a marked enlargement of the cord as seen externally, but it may extend downwards and upwards even into the medulla. Transversely it may spread so as to involve the horns of gray matter and the posterior and lateral white columns. The new tissue is liable to break down in its centre, partly from want of proper organisation, partly from hæmorrhages which may take place into it, and thus there is formed, in the cervical region particularly, a canal—placed behind the normal canal—with irregular borders, and not lined with ciliated epithelium.

FIG. 22.—Lumbar enlargement from a case of tumour of the spinal cord, illustrating one of the varieties of cavity formation or *syringomyelia*. A star-shaped cavity is shown in the posterior column and the crossed pyramidal tracts show some degeneration of the nerve fibres.

The TUMOURS that involve the cord may be of very various kinds—fatty, fibrous, myxomata, gliomata, sarcomata, carcinomata, psammomata. Echinococci, as a rarity, has been found within the spinal canal. Tubercle and syphilis have already been mentioned. The microscopical anatomy of spinal new growths need not detain us, since it does not differ from that of such growths elsewhere. The importance of innocent tumours (unfortunately not the most common variety), which arise outside the cord itself, is mostly derived from their pressure effects, and from the possibility, which in one or two instances has been demonstrated, of localising and removing them. As to malignant growths, allusion has been made to the destruction of the nerve roots and the cord that takes place in cancer of the vertebræ. And a similar transverse myelitis, partly from pressure, partly from infiltration, is no doubt the

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commonest result of all malignant disease, whether this start from bones, nerve roots, or meninges. There will be still earlier disorganisation of the cord in those rarer cases where the new growth originates in the substance of the cord itself. It may be just mentioned, however, that small malignant growths have been found upon the inner surface of the dura, whose effects, if any, have been by way of pressure, and that sarcomatous tumours have been described, which extend a long way in the meninges, enwrapping and compressing the cord rather than destroying it.

General Symptomatology

In order to avoid repetition, it will be convenient, before describing the various diseases of the cord, to discuss certain general symptoms which frequently present themselves, and depend rather on the locality of the lesion and the function of the part involved than on the nature of the particular morbid process.

Two main types of **motor paralysis** may be distinguished—the spastic and the flaccid.

Spastic paralysis occurs in connection with destructive disease of the upper motor neurone, represented in the spinal cord by the pyramidal tracts, and particularly in connection with chronic degeneration of these tracts. Flaccid paralysis, on the other hand, points to destructive disease of the lower motor neurone, represented in the spinal cord by the large cells of the anterior gray cornua, and by the fibres which arise from them and pass out as the anterior nerve roots.

Spastic paralysis is characterised by an abnormal increase of muscular tonus, which in its slighter or earlier stages manifests itself mainly by increase of tendon reactions. Thus the knee jerks become exaggerated, and ankle clonus appears. The muscles do not waste; there is no change in their electrical reactions. In more advanced stages the muscles enter into a state of chronic contraction or "contracture." The limbs become rigid; the position which they assume depends on the prēponderating action of certain strong groups of muscles. Commonly, but not invariably, the lower limbs are extended at the hips and knees, and adducted at the hips; the upper limbs adducted at the shoulder, semiflexed at the elbow; the hand semipronated and the fingers flexed. As a stage in advancing rigidity, we sometimes notice that the lower limbs, not yet permanently stiff, occasionally draw themselves up spontaneously, or become rigid directly they are manipulated.

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The gait of a patient with spastic paralysis of the lower limbs, supposing that he can still walk, is peculiar. The leg that is coming forward is brought up slowly and with difficulty, with very little flexion either at the knee or ankle, so that the toes drag along the ground. To help in clearing the toes, there may be a certain tilting of the pelvis towards the opposite side, or a movement of circumduction at the hip joint. The boots wear out at the toes. This laborious dragging gait may be profitably contrasted with the "high-stepping" gait of a patient suffering from paralysis of the extensors of the toes, or with the flourishing of the feet with stamping of the heels that is sometimes seen in advanced locomotor ataxy.

In *flaccid paralysis*, on the other hand (whether it be produced by destruction of the anterior cornual cells, or destruction of the motor nerves or nerve roots, with consequent separation of the muscles from these nutrient cells), there is from the beginning loss of tonus and flaccidity of the muscle. Wasting soon begins, which may proceed to complete atrophy. While this is proceeding, or indeed before it is noticeable, characteristic changes may be found in the electrical reactions called "reaction of degeneration." When the faradic current is applied (to the muscle or nerve trunk supplying it), the muscle no longer contracts as normally it should do. The same defect is noticed when the galvanic current is applied to the nerve. When the latter is applied to the muscle, the muscle contracts, indeed may do so more readily than normal, but there are peculiarities in its action, namely that (α) the contraction of the muscle which constitutes the response to the make or break of the current is somewhat sluggish and prolonged, not a quick twitch; (β) the action of the anode may be as marked as, or more marked than, the action of the kathode. The tendon reactions, so far as they depend upon action of the paralysed muscles, disappear, and the limb is from first to last limp, not rigid.

There are some apparent exceptions to the above statements. Thus in some cases of hemiplegia, where the disease is seated in the upper neurone, we may find wasting of muscle. Such wasting, however, is less extreme, and is never accompanied by electrical reaction of degeneration. Again, in some forms of muscular atrophy which apparently originate in the muscle rather than in their nutrient nerve cells (myopathic atrophies), there may be extreme wasting but no reaction of degeneration. Still these two types of paralysis may be accepted on the whole, viz. spastic paralysis associated with degeneration of the upper neurone, flaccid paralysis with that of the lower.

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Concerning **sensory paralysis**, the most definite statement we can make is that when the cord is completely severed at any one level sensory paralysis, as well as motor, ensues in all parts of the body below. But the dorsal part of the cord is most liable to such severance, and here, from the disposition and innervation of the trunk muscles, it is difficult to delimit the motor paralysis, whereas the line of sensory paralysis can be more easily drawn. From this line we can infer the level at which the cord is severed: an inference, the exactness of which depends on a knowledge of the functions of the various segments of the cord, to which we shall refer again presently.

There are certain rare cases characterised by motor paralysis of all parts upon one side of the body below a given level, and sensory paralysis of all parts below the same level on the other. This is known as *hemiparaplegia*, and has been explained by the assumption that the sensory fibres, after entering the cord *viâ* the posterior nerve roots, cross to the opposite side before proceeding upwards towards the brain; so that a lesion involving one lateral half of the cord would cut all motor fibres going downwards to the same side, and all sensory fibres coming upwards from the other. Against this interpretation of the facts, however, difficulties have been urged.

"Dissociation" in sensory paralysis means that while one form of sensation is annulled, others remain intact. In syringomyelia particularly it may be noted that while tactile sensation is at first unaffected, thermal sense and pain sense are paralysed. This is held to mean that the tracts for the latter class of sensation run separately from those of the first, and probably in the neighbourhood of the central canal. Such dissociation is sometimes seen in other diseases, for example tabes.

Defective combination of muscular contractions produces an irregularity of action that is called *ataxia or inco-ordination*. Such defect may result from disease of very various parts of the nervous system—intracranial, spinal, or even peripheral. When spinal, the disease is situated in the posterior columns of the cord, or in the posterior nerve roots, and of such inco-ordination tabes or "loco-motor ataxia" supplies the commonest and most typical example.

Anomalies of reflex action supply, as might be expected, most valuable evidences of spinal disease. Alteration in the *tendon reactions* are often of very definite significance, and in the knee-jerk particularly, because this is a phenomenon always present in health and easy to observe. Mention has already been made of the principal condition under which the tendon reactions become

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increased, namely, chronic degeneration of the pyramidal tracts, or gradually increasing pressure upon them. But in certain cases where the dorsal cord has been completely severed, whether this be due to injury or to a total transverse myelitis, the knee-jerk is permanently abolished, and the lower limbs remain lax, not rigid, although descending degeneration may have begun in the pyramidal tracts below the lesion. Doubtless the tendon reactions become exaggerated also when the irritability of the anterior cornual cells becomes abnormally increased; of this strychnia poisoning and tetanus supply examples; but this is a matter of functional disturbance rather than of structural disease. They may be exaggerated also in hysteria and neurasthenia, diseases of which we do not yet know the full significance. Further, they vary much in different healthy individuals. Ankle clonus is but a special manifestation of increased tendon reaction, yet it has this peculiarity that it rarely occurs in healthy people; indeed it is a rule with few exceptions, that permanent typical ankle clonus is as much a sign of structural disease as is permanent absence of knee-jerk.

Absence of knee-jerk points to disease of the reflex arc, on the integrity of which this phenomenon depends, the spinal part of which is situated in the third and fourth lumbar segments, and the peripheral part in the nerve roots proceeding from them. When the disease is in the afferent part of this arc, *i.e.* when it does not involve the anterior cornual cells or anterior nerve roots, the knee-jerk is absent but there is no paralysis, muscular wasting, nor electrical reaction of degeneration; when the disease does involve these parts (involves, that is to say, the lower motor neurone), then these symptoms of atrophic paralysis appear. It must be remembered also that the knee-jerk may be annulled by influences from above the reflex arc; this may happen in some cases of intracranial disease, or from spinal disease completely cutting off the lower centres from the cerebral, as we have just remarked. (See page 11.)

The various "*superficial*" or "*skin*" *reflexes* have not hitherto afforded us so much help in diagnosis as we might expect. This is mainly due to the fact that in health they vary much, and, indeed, are often absent, so that their absence is no conclusive evidence of disease. This much we may say, that when a given reflex is present the arc on which it depends (*vide* table, p. 210) has not been destroyed at any point of its course. Nevertheless, it would seem that certain recent studies upon the plantar reflex are likely to give some valuable clinical results. The normal reflex produced by tickling the sole

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(in addition to certain movements at the hip and ankle) consists of *flexion* of the toes. But where there is disease affecting the pyramidal tract *extension* of the toes, and of the great toe in particular, may take the place of flexion, and it is held that this "extensor type" of plantar reflex points as a rule to organic disease affecting the pyramidal tract (in brain or cord). The converse is not necessarily true, for the absence of this phenomenon does not absolutely negative the presence of such disease.

Under the head of *organic reflex actions*, those of most clinical importance are the actions of the bladder, rectum, and pupil. *Micturition* is primarily a spinal reflex action, but it may be materially modified by cerebral influences. Reference may be made here to such of the commoner affections of micturition as are distinctly due to spinal disease. (i.) If a transverse lesion in the dorsal region of the cord cuts the communication between the brain and the reflex centre for micturition, this latter remaining in itself intact. When this happens suddenly, retention of urine with distension of the bladder is usually the first result. This has, of course, to be relieved with the catheter. After a time, however, the centre may acquire independent power, and a condition known as "reflex incontinence" results, that is to say, the bladder empties itself at intervals without effort, or perhaps without knowledge, on the part of the patient. (ii.) If the centre for micturition in the second sacral segment be destroyed the sphincter and the bladder walls are then paralysed, and neither voluntary nor reflex emptying of the bladder is possible. In consequence of mechanical obstacles at the outlet the organ first distends, and then the urine gradually dribbles from it, "overflow incontinence," as it may be termed. (iii.) Micturition may be affected in less definite ways, either when the lesion of the cord above or of the centre is incomplete, or when the reflex is disturbed by disease of its afferent fibres, as would appear to be the case in early tabes. In this last-mentioned condition "precipitate micturition," in which the patient must hurry to pass water directly the desire takes him, is not uncommon.

It is probable that the mechanism of *defæcatiòn* is very like that of micturition, but it is more likely to be complicated by accidental conditions, such as the hardness or fluidity of the fæces, the peristaltic action of the upper bowels, the integrity or paralysis of the abdominal muscles and diaphragm.

The commonest abnormality of the *pupil reflex*, *i.e.* absence of contraction under light, which occurs in tabes and general paralysis with great frequency, is due to lesion in the pons, and is therefore

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not a spinal symptom, anatomically speaking. As to dilatation, the fibres which dilate the pupil run down from the brain in the lateral columns of the cord, and leave the cord *viâ* the second dorsal nerve root to join the sympathetic. Consequently a small pupil, which does not dilate readily when shaded, may be one indication of disease involving this nerve root, or the deeper parts of the cord above it.

We may mention here some of the **disturbances of nutrition** which present themselves in the course of spinal cord disease. Disease of joints and of bone, peculiar in character, occurs in tabes, in general paralysis, and in syringomyelia; whitlows, bullæ upon the skin in syringomyelia; perforating ulcer of the foot is tolerably common in tabes. But we cannot as yet refer these symptoms to lesion of a particular part of the cord in the same way in which we definitely connect muscular atrophy with lesion of the anterior cornual cells. Bed-sores constitute the nutritional disturbance of most practical importance, and these occur with the greatest readiness in cases where a large transverse area of the cord is diseased, as in a transverse myelitis, affecting afferent and efferent tracts and gray matter. The severe form of cystitis which follows myelitis of the lumbar enlargement is the analogue on the mucous membrane of the bed-sore on the skin; both are doubtless due partly to defective innervation, partly to local irritation. The typical position of a bed-sore in spinal disease is over the sacrum. (See "Trophoneuroses," p. 369.)

Segmental Symptoms.—A knowledge of the parts supplied from the various spinal segments is often most useful in clinical investigations. These parts are exhibited in the table, p. 210. But it must be remembered that on this head our knowledge is incomplete, inasmuch as the statements of different authorities disagree in some details, and, secondly, that in consequence of the "overlap," already noticed (p. 14), in the muscular and skin supply from adjacent spinal segments, absolutely accurate localisation of spinal disease from peripheral symptoms may not always be possible. Nevertheless, broadly speaking, the various segments contain the nutritive cells for certain muscles, the centres for certain reflexes, and the afferent and efferent paths flowing through the corresponding nerve roots. Hence the annulling of such reflexes, atrophic paralysis of such muscles, and sensory disturbances in certain skin districts may be spoken of as "segmental symptoms." Such segmental symptoms do not differ materially from the symptoms caused by disease of the corresponding nerve roots

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("root symptoms," in the strict sense). Yet it is sometimes important to know whether the nerve roots or the cord itself be primarily implicated, and the following considerations perhaps may be of help:—(1) Prolonged neuralgic pains, limited to a particular segmental district, point to nerve-root lesion; (2) if segmental symptoms, whether motor or sensory, be accompanied from the first by paraplegia the cord is more likely to be primarily involved than in cases where paraplegia follows late (as a result of compression). [This does not apply to cases of progressive muscular atrophy where the anterior cornual cells are specially picked out, nor yet perhaps to most cases of Pott's disease, in which segmental symptoms are often slight.]

But, at any rate, the localisation, as to level, of spinal lesions, whether they affect the cord itself or the nerve roots originating from it, depends largely upon the class of symptoms referred to, namely, the particular distribution of pain, of anæsthesia, of atrophic muscular paralysis, and the interruption of particular reflex arcs.

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DISEASES OF THE SPINAL MEMBRANES

Meningeal Hæmorrhage

Hæmorrhage within the spinal meninges is so rare as a primary disease that it hardly requires description, further than to say that the symptoms are likely to resemble those of an acute spinal meningitis, but to come on still more suddenly, and that, whereas in a meningitis there would be fever from the first, in a hæmorrhage this would not be so.

Acute Spinal Meningitis

Symptoms.—Acute meningitis affecting the pia and arachnoid of the cord in its whole length is generally a part of a cerebro-spinal meningitis. The constitutional symptoms of this condition need not be repeated; of local symptoms the cerebral usually predominate, but sometimes only the spinal are in evidence. These are pain in the back, at the nucha and along the spine, which is aggravated by movement either of these parts or of the limbs; tenderness of the spine brought out either by pressure or by the application of a hot sponge. Pain is felt too in the limbs and around the body (doubtless due to the implication of sensory nerve roots), often accompanied by extreme hyperalgesia to touch, and sometimes by vasomotor disturbances causing a "tache," or even cutaneous eruptions, such as herpes. A second feature is rigidity; the head is drawn back, the spine often arched, the legs drawn up and flexed at the knees. Probably such rigidity is due to active muscular contraction, resulting either from irritation of the nerve roots or from semi-voluntary attempt to avoid the pain of movements, and is not analogous to the contractures of old spinal disease. If, while the hip-joint is semi-flexed, attempts at passive extension of the knee-joint be made, resistance is experienced from contraction of the flexors of the knee. The disease may run a rapid course, and the patient may perish in a few days or less from implication of the respiratory or cardiac movements, or still more commonly from cerebral causes. Some cases are more prolonged, and in these paralysis, not usually an early symptom, may appear. This may be

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due either to implication of the cord itself, or to pressure upon and infiltration of the motor-nerve roots. Consequently it is often of the atrophic type. Anæsthesia, of very varied distribution, may arise for the same reason, and paralysis of the bladder and rectum. The knee-jerks disappear as the nerve roots become involved, whatever their condition may have been in the earlier stages.

The **prognosis** is grave; acute and severe cases generally end rapidly, and even in cases that recover there remains the risk of permanent paralysis from damage done to the cord or nerve roots.

The **diagnosis** depends largely on the symptoms of irritation, viz. pain, tenderness, and rigidity with fever. In these respects meningitis may be to a certain extent simulated by less serious conditions, such as rheumatism, influenza, hysteria. On the other hand, it is conceivable that, as in some of the worst cases of cerebrospinal meningitis, coma is from the first the leading symptom. So spinal meningitis might manifest itself mainly by paralysis, in which case it would be difficult to distinguish from such conditions as acute ascending myelitis, Landry's disease, or a rapidly advancing multiple neuritis. Exploratory puncture of the theca spinalis in the lumbar region has been recommended, firstly, for the diagnosis of exudative meningitis from such other diseases; secondly, for the diagnosis of purulent from tuberculous meningitis, by examination of the nature of the exudation.

Other Forms of Spinal Meningitis

Just as in acute purulent meningitis, so in the less acute *tuberculous meningitis*, it is rare for the spinal symptoms to predominate. Still this may sometimes happen, and the possibility of a tuberculous meningitis should not be forgotten, when obscure paralytic symptoms of spinal origin arise in a patient whose age and antecedents render him liable to this form of tuberculosis.

Subacute meningitis, of *syphilitic* origin, is common, but the symptoms generally point to local, or at most disseminated lesions rather than diffuse disease, and to implication of the cord as well; it will be best considered under the head of transverse myelitis.

A chronic diffuse meningitis, such as may be due to alcohol or to Bright's disease, is difficult to diagnose in the cerebral membranes, and in the spinal is probably impossible.

PACHY-MENINGITIS CERVICALIS HYPERTROPHICA reveals itself, according to Charcot, first by pains in the neck, back of the head,

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and down the arms, and by numbness, anæsthesia, and sometimes cutaneous eruptions in the upper limbs. Next comes atrophy of the intrinsic muscles of the hands, and of the flexors of the fingers and wrists. Since the extensors escape, a peculiar position of extension of the wrists and fingers results, which is sometimes called "the preacher's hand." All these are root symptoms, caused first by irritation, secondly by paralysis of the cervico-dorsal nerve roots. Finally, paraplegia may follow from compression of the cord. The disease is thus clinically a cervical paraplegia having a somewhat special localisation, and it does not appear that the symptoms differ from those produced by any chronic meningitis occupying the same level.

Treatment.—For an acute purulent meningitis little can be done, save absolute rest, careful feeding and nursing. Dry cupping to the spine, or inunctions of mercury along the spine have been recommended in the acute stage. It has been held, too, that morphia may do something to control the disease, as it certainly may be required to relieve the distress. Paralytic sequelæ are best treated by iodide of potassium, and by the local application of electricity and massage. The treatment of tuberculous meningitis is eminently unsatisfactory. We need hardly say that where there is the least probability that a meningitis depends upon syphilis, mercury and iodides should be given and pushed as required. These same drugs, and more particularly iodides, are commonly given in the more chronic forms of meningitis.

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DISEASES OF THE SUBSTANCE OF THE SPINAL CORD

A. NON-SYSTEM DISEASES OF THE CORD

(1) LOCAL. ACUTE ANTERIOR POLIOMYELITIS; HÆMORRHAGE INTO THE CORD; TRANSVERSE MYELITIS (PARAPLEGIA).
(2) DIFFUSE. ACUTE DISSEMINATED MYELITIS; DISSEMINATED SCLEROSIS.
(3) SYRINGOMYELIA; HYDROMYELIA.
(4) TUMOURS OF THE CORD.

ACUTE ANTERIOR POLIOMYELITIS

Acute anterior poliomyelitis, or infantile spinal paralysis, is an acute disease, either inflammatory or degenerative, affecting a limited portion of the anterior gray cornua (see p. 160).

Between the ages of one and five years it is quite common; in youth and early adult life it occurs, but with far less frequency; in old age it is practically unknown.

Its **etiology** is not certainly known, but it is commonly thought to be due to an infection by micro-organisms. This view is based on its mode of onset, which resembles that of an infectious disease, on the fact that it has a certain seasonal prevalency (being commonest between June and September), that it has been known to occur in epidemic form, and that we are ignorant of any other likely mode of causation.

Symptoms.—The child, who more often than not has been previously healthy and robust, suddenly evinces signs of acute *constitutional disturbance*, such as feverishness, vomiting, and sometimes convulsions, and while thus laid up paralysis sets in, of one or more limbs, or of limbs and trunk, so that the patient cannot sit up. This will probably occur within the first day or two of the illness, but in mild cases may not be noticed while the child is still in bed. There may be pain and tenderness in the paralysed parts at first. The constitutional symptoms, however, may be of varying degrees of severity, and sometimes are so far wanting that there is little to attract attention save the loss of power.

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The *paralysis* has the following characters: it affects the limbs principally, one or more; the face, tongue, eyes, and sphincters are rarely, if ever, attacked; there is no sensory affection save perhaps initial pain and tenderness. The maximum paralysis is evident from the first; after remaining stationary for a short time it begins to improve and to limit itself to the parts that are to remain permanently paralysed. The eventual distribution varies much in different cases; common forms are both legs, one leg, one arm, arm and leg of one side, or groups of muscles in one limb, the grouping of such paralysed muscles following the arrangement of the nuclei in the spinal cord.

The paralysis is of the atrophic type; the muscles are flaccid from the first; changes in their electrical reactions begin within a fortnight, typical reaction of degeneration often occurring; and before long the characteristic atrophy of muscle begins to show itself. This may become extreme in the permanently affected parts. In severe and long-standing cases the bones do not grow properly, so that the affected limb becomes shorter than its fellow. The skin, too, becomes cold, and in winter it exhibits a blue congested appearance, and is subject to chilblains. Lastly, deformities and particularly various kinds of talipes are gradually produced, from the unbalanced action, and eventually the permanent contraction, of the non-paralysed muscles.

Diagnosis.—Infantile paralysis due to cerebral lesion, a fairly common disease, may have an onset resembling that of acute anterior poliomyelitis. The distinctions are as follows — the paralysis of cerebral disease is strictly hemiplegic in distribution; tongue, face, and speech often suffer; in the spinal disease the distribution is not usually hemiplegic, even in the limbs, and the tongue, face, and speech are unaffected. In the cerebral disease, the paralysis is spastic in type, the tendon reactions are increased, the electrical reactions are preserved (at most, there is diminished electro-contractility); in the spinal, the paralysis is atrophic, and the tendon reactions (so far as they are subserved by paralysed muscles) disappear. Lastly, as a late result of the cerebral disease we may see athetosis, or hemiplegic contracture; of the spinal, deformities from partial paralysis of a limb. Atrophic paralysis in childhood, commencing acutely, may conceivably be due also to peripheral neuritis, but the distribution will in this case be different, and there will be some sensory paralysis; or to hæmorrhage into the cord, but this is so rare that we need hardly consider it here.

Prognosis.—The disease is very rarely fatal, so far as we know.

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But it is possible that a case, terminating fatally at an early stage, might never be recognised for the want of distinctive paralytic symptoms. There are three stages in the disease—(α) the onset, (β) a stationary stage, (γ) the stage of regression or improvement. At the outset it is impossible to say how far improvement will go. Anatomically indeed we know that, where nerve cells are irretrievably destroyed, the muscles that are dependent upon them must perish for good, but that, if the cells be only affected temporarily, the muscles will recover. But in practice we can only judge of this by symptoms; partly by the rate at which improvement (when once set in) has been and is taking place, partly by the electrical reactions of the affected muscles. Where regression of the paralysis has begun early, and is still proceeding, probably there will be further improvement, though complete recovery is very rare. Muscles which after ten days or so still retain their contractility to faradism will probably recover, neither does the appearance of reaction of degeneration preclude recovery of muscles. On the other hand, if the paralysis have remained stationary for many months, and if the paralysed muscle refuse to contract under any form of electrical stimulation, the prognosis is bad, and all the more so if careful and continuous treatment have made no difference to the condition.

Treatment.—Little can be done during the stage of onset save keeping the child absolutely at rest in bed. Mild counter-irritation, or dry cupping, to the spine has been recommended. After some two or three weeks, treatment of the paralysis may be begun. We cannot pretend to replace destroyed nerve cells, or obviate the paralysis of muscles innervated from them; but we aim at maintaining the nutrition and hastening the recovery of muscles whose centres may have suffered less severely. For this massage and passive movements should be employed, and electrical applications, best of the constant current, inasmuch as the paralysed muscles rarely react to faradism. As voluntary power returns, the child should be encouraged to move and use the limb. The limb should be carefully kept warm; a thick glove or stocking or a layer of cotton wool should be worn in the winter. It may be sponged well with tepid water, and then briskly rubbed to quicken the circulation. As medicine, strychnine has been employed with a view of influencing the paralysis; cod-liver oil and iron may be necessary to maintain the general health. Perseverance and regularity must be the guiding principles of treatment; eminent authorities hold that much may thus be done even for the most

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unpromising cases. The deformities that result in old cases have to be met by surgical means, and the application of mechanical boots and supports. An ingenious plan of treatment has lately been devised, which consists in attaching portions of the tendons of non-paralysed muscles to those tendons whose muscles are hopelessly paralysed.

HÆMORRHAGE INTO THE CORD

Hæmorrhage into the substance of the spinal cord is of very rare occurrence as a primary disease. Sudden onset of paralysis, accompanied with pain, appear to be the leading features. Obviously the details of the paralysis must depend upon the situation of the lesion. But it is said that the gray matter suffers most severely, whence we should expect an atrophic paralysis to result, at any rate if the hæmorrhage be in the lumbar or cervical region. Hence a confusion with an acute anterior poliomyelitis might conceivably arise. If it be in the dorsal region, we should probably be unable to distinguish it from an acute transverse myelitis.

TRANSVERSE MYELITIS

One of the commonest forms of local spinal disease is that known as "transverse myelitis." It has been explained that this means disease extending more or less completely across the whole section of the cord, but involving only a small length of it—in short, segmental. The disease may have begun in the cord itself, or be secondary to affections of the bones or meninges. The symptoms are of two kinds—paraplegic and segmental; the relative importance of these two groups will vary according to the particular segment which is involved, and according to the degree in which the primary disease involves the nerve roots or no.

OF THE DORSAL REGION

The dorsal region is the commonest seat of transverse myelitis.

The **symptoms** then constitute a typical *paraplegia*, consisting mainly in loss of voluntary power in the lower limbs, loss of sensation in all parts below the level of the lesion, loss of control over micturition and defæcation. The completeness of such

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paraplegia will depend upon the completeness with which conduction in the cord is interrupted; in incomplete lesions, the relative preponderance, *inter se*, of these symptoms may vary, but usually the motor paralysis is in excess of the loss of sensation, and of the sphincter affection. On the other hand, the level of the anæsthesia, when this can be distinctly defined, gives the most definite indication of the level of the lesion; otherwise *segmental symptoms* are at a minimum. Girdle-pain, *i.e.* an oppressive sensation like a cord constricting the body, may indeed be present; and further, if the lesion be one which has specially attacked the posterior nerve roots, severe lancinating pains may have been felt in the area of their distribution.

The paralysis of the lower limbs is at first characterised by simple loss of power, but before long it begins to assume a spastic type; the knee-jerks become exaggerated, ankle clonus appears, the limbs draw up spontaneously or when manipulated, and eventually become rigid. Permanent spasticity is no doubt due to secondary descending degeneration of the pyramidal tracts. The exception, already mentioned, must be remembered, that when a whole cross section of the cord is destroyed (complete transverse myelitis), the knee-jerks are absent, and remain absent, in spite of descending degeneration. The converse statement, that if the knee-jerks be absent a whole section of the cord is destroyed, is probably not always true, at any rate in the earliest stages of a paraplegia; allowances must be made for temporary influences of the lesion.

Retention of urine is usually an early result of a severe dorsal myelitis, and this must be relieved by the catheter. Such retention may, however, give way to "reflex incontinence." There is commonly retention of fæces, but this may be varied by involuntary and unconscious discharges. Priapism is often present. Bed-sores are much to be dreaded. These appear on parts subject to pressure, the sacrum particularly, the heels also, and the ankles when they get pressed together from spasm of the legs. Irritation from urine or fæces, in badly nursed cases, is also a predisposing factor. But the nervous lesion is no doubt the most important element in their production, at least in the form known as "acute decubitus." This kind of sore develops with an alarming rapidity, and in spite of all precautions. A patch of erythema is discovered, which assumes a dark colour in its centre; blebs of fluid may form on this, and before long deep-seated and extensive sloughing of tissues sets in.

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OF THE CERVICAL, LUMBAR, OR SACRAL REGIONS

If the myelitis affect the cervical or lumbar enlargements, segmental symptoms assume more importance. Thus in disease of the cervical enlargement ("cervical paraplegia," as it may be called clinically) there is paraplegia such as we have described, with the addition that the intercostal muscles are paralysed. But there is also atrophic paralysis and anæsthesia in the upper limbs, and from the distribution of this we may be enabled to localise the segment of the cord which is diseased. In disease of the sacral region, involving the centres for the bladder and rectum, there is distension of the bladder, with overflow, incontinence and probably cystitis, and the sphincter ani is paralysed. But there is not necessarily any paralysis of the lower limbs, and anæsthesia may be limited to the perinæum and adjacent parts. If the lumbar enlargement be affected at a higher level, there will be atrophic paralysis and anæsthesia of the lower limbs in the areas supplied from the level of the disease, and the knee-jerks will be abolished.

The rapidity of onset in cases of paraplegia may vary greatly, according to the nature of the process at work. In some, rather uncommon, cases the onset is sudden, and probably there is then a vascular obstruction due either to syphilitic disease, or to causes upon which we cannot certainly pronounce. Or after prodromata, such as tingling, numbness, and other paræsthesiæ in the legs, a paralysis may arise which reaches its maximum in a day or two. Or the onset may be spread over some weeks. Lastly, there are cases in which the symptoms appear very slowly, in one limb perhaps before the other, and gradually settle down into a typical paraplegia. These, in our ignorance of their exact causation, we are wont to call chronic transverse myelitis; some of them eventually turn out to be cases of disseminated sclerosis.

When the onset of a paraplegia is preceded by marked "root symptoms," such as pains and paræsthesia followed by anæsthesia in the distribution of certain nerve roots, coupled, it may be, with muscular atrophy of a corresponding distribution, it is likely that the morbid process originates outside the cord in the bones, meninges, or in the nerve roots themselves.

The course of a paraplegia, once established, must necessarily depend (1) upon the nature of the morbid process which originated it, whether this be progressive or tractable; (2) upon the complete-

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ness with which the nervous tissue has been destroyed. That is to say, when the cord either has been or is likely to be completely cut across, improvement cannot be expected. Such extreme cases are likely to be cut short by the complications of cystitis or bed-sores without becoming truly chronic. Most cases of chronic paraplegia have either been incomplete from the onset, or have made some improvement within a moderate length of time. Sensation probably returns, and a certain amount of control over the sphincters, but a spastic paralysis of the legs, of a greater or less degree, remains permanently. This may be reasonably ascribed to a descending degeneration of the pyramidal tracts, started by the original transverse lesion. Indeed, such cases are sometimes spoken of as "lateral sclerosis," but this is incorrect if it be thereby implied that such lateral sclerosis was the original disease.

Certain Common Causes of Paraplegia

It is desirable now to consider the **symptoms** of transverse myelitis in relation to those diseases which are the commonest cause of it, although this plan may lead somewhat beyond the limits of strictly local disease of the cord.

It is very often associated with CARIES OF THE VERTEBRÆ (Pott's disease). Such vertebral disease may be revealed by deformity or immobility of the spinal column: or the nervous symptoms may be the first to appear. In the latter case we can only conjecture their cause; our conjectures are based upon the age and constitution of the patient, the absence of other causes, and the mode of onset of the paralysis, which is usually gradual. If the disease is in the dorsal region, as is most frequent, the first complaint is usually of a weakness of the legs. Although the bulk of the disease is meningeal, root symptoms are not usually a marked feature. Pain in particular is not severe. The knee-jerks are exaggerated from the first, and ankle clonus develops early; rigidity will develop if the case gets worse. The other paraplegic symptoms may follow, namely, sphincter trouble, and anæsthesia of the lower limbs. Marked paraplegic anæsthesia is thought to indicate grave interference with the cord, either by pressure or infiltration, and the prognosis is therefore the worse.

The course of the disease varies considerably. The symptoms may get worse week by week, or progress much more slowly, or remain stationary. Again, under favourable circumstances, even severe cases may improve, and the patient get practically well.

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Such variations can be accounted for by the want of uniformity in the progress of the caries; there may be increase of pressure from meningeal deposit, from formation of abscesses, or displacement of a vertebra, or increased spread of disease into the cord itself, or, on the other hand, the tuberculous tissue may shrivel and become obsolete before the cord has been hopelessly damaged.

When the disease attacks the cervical region muscular atrophy in the upper limbs may be a prominent feature, and in the rare cases where it affects the lumbar region, the knee-jerks may be absent, instead of being increased.

Treatment may be based on three plans, or rather on a judicious combination of them.

(1) Restoration of the general health by feeding, tonics, iodides, cod-liver oil, in the hope that the local disease will thereby undergo involution.

(2) Rest. Prolonged confinement to bed may have the effect of relieving spasm of the limbs, and of promoting quiescence in the local disease. This latter object is also sought by the application of spinal supports and jackets; but for progressive cases there is no efficient substitute for rest in bed.

(3) Relief by surgical operation.

It may be possible, after laminectomy, to find collections of pus that can be evacuated, or diseased bone that can be scraped; and thus pressure may be relieved, or the diseased parts put in a better condition for healing.

We have already mentioned that SYPHILIS may affect the cord in many ways. We have not space to describe the various symptoms to which it may give rise, in relation to the structures which it affects; nor indeed would this be profitable, seeing that the lesions are often multiple. A combination of symptoms that points to more lesions than one (such as a paraplegia preceded or followed by a hemiplegia or by ocular paralysis) is in itself somewhat suggestive of syphilis. Taking the subject of paraplegia only, we have seen that this may be caused by a local meningo-myelitis, or by arterial disease leading to softening, or more probably by a combination of the two; or a gummatous tumour may compress cr infiltrate the cord. The onset of this paraplegia may consequently be gradual, with such prodromata as numbness, tingling, and occasional failures of power, or acute. Pain is not the rule, but may be present if the nerve roots are gravely involved. As regards the paraplegic symptoms themselves, it is reasonable to suppose that these must depend upon the completeness of the transverse lesion,

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or if this be incomplete upon the degree in which it affects the various structures of the cord, rather than on the pathological nature of the lesion; nevertheless it is maintained by Erb that a certain grouping of the symptoms is suggestive that the paraplegia is syphilitic. This combination is as follows: incompleteness of motor paralysis (except at the early stage); very marked increase of tendon reactions, yet without much rigidity; sensory symptoms, such as numbness and paræsthesia, yet no marked anæsthesia (except at the early stage); bladder symptoms marked, considering the incompleteness of the motor paralysis.

The course of a syphilitic paraplegia is necessarily connected with the subject of **treatment.** In all cases of central nervous lesion which can be conceivably due to syphilis directly, it is now a routine rule to administer anti-syphilitic remedies promptly and sufficiently. This is the more necessary in spinal disease, since the area of the cord is so small, and destruction of its nervous tissues is irreparable. On the other hand, we know that gummatous growth may be easily removed by anti-syphilitic medicines: hence much depends upon the promptness of the treatment. The whole question of **prognosis** will depend upon how much nervous tissue has been destroyed, and this unfortunately we are hardly in a position to know in any given case till we have watched the effect of our remedies.

A NEW GROWTH affecting the spinal cord will sooner or later cause paraplegia. The symptoms which precede this consummation necessarily vary according to the character and primary position of the growth. Pain is one of the most constant symptoms. There may be local pain in the back, aching, boring, or stabbing in character; this is often aggravated by movement of the back, or of the limbs, and the vertebræ may be tender. There may be lancinating neuralgic pains, round the trunk or down the limbs, due to irritation of nerve roots, likely to be followed by anæsthesia and atrophic paralysis of segmental distribution. Or there may be pains in the lower limbs and parts below the seat of lesion, probably due to the irritation of the intra-spinal fibres which conduct pain sensations from the periphery to the brain. Again, in some cases the diagnosis may be greatly aided by the appearance of a local tumour of the spine, or the nature of the lesion may be indicated, in an otherwise doubtful case, by the history or presence of new growths elsewhere. The paraplegia itself will probably develop gradually, being in most cases due to compression, but it is quite conceivable that where a secondary myelitis, or a softening from arterial

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thrombus takes place, its onset may be acute or even sudden. Where the growth begins within the cord itself we should expect the paraplegia to be of early occurrence, perhaps the first symptom. Growths springing from the meninges may be expected to attack the nerve roots, and hence "root symptoms" will be a valuable and early sign. We must here again allude to the fact that small growths affecting one nerve root have been during life successfully diagnosed and removed in one or two instances. The combination of pain and paralysis caused by cancer of the vertebræ has led to the designation "paraplegia dolorosa."

It should be remembered that another possible cause of paraplegia preceded by pain is ANEURISM OF THE AORTA.

Only the principal diseases which give rise to paraplegia have been referred to; but this symptom may of course arise from solution of continuity in the spinal cord from whatever cause. On the other hand, loss of power, and of sensation in the lower limbs, may be due to other causes than spinal disease, such as peripheral neuritis and hysteria. These subjects will be treated of in other sections.

ACUTE MYELITIS, DIFFUSE OR DISSEMINATED

Acute or subacute myelitis, which is not limited in area, is on the whole uncommon. Sometimes myelitis spreads upwards from the site of an injury to the cord, as in the case of fracture to the vertebræ. To the original paraplegia there is then added, day by day, loss of pain and sensation in the parts above the level of the fracture (see p. 159).

Symptoms.—Acute ascending myelitis sometimes sets in without such definite cause. After some trivial injury or strain to the back, after exposure to cold, or without assignable cause, pains in the back, and numbness or paræsthesia of the legs begin. These are soon followed by loss of power, and presently a complete paraplegia of motion and sensation is established. The legs are flaccid, the knee-jerks absent, loss of muscular electro-contractility follows in a week or so. The bladder and rectum become paralysed; cystitis and bed-sores very frequently set in. The paralysis spreads upwards, involving at last the respiratory muscles. We should expect in such a case to find post-mortem a disorganisation of the cord either general and diffuse, or involving numerous scattered areas of it, with which might possibly be associated an inflammation of the

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meninges. Probably an infective process is at work, and micro-organisms have indeed been found in such cases. Acute ascending myelitis is distinguished from acute ascending paralysis, otherwise known as Landry's disease (see p. 221), by the fact that in the latter disease there is no definite anæsthesia, the sphincters are not commonly paralysed, and the electrical irritability of the muscles is retained throughout. It is, however, quite conceivable that in the early stages, or the less typical forms of myelitis, these distinctions might fail us.

The symptoms of an acute or subacute disseminated myelitis, in which numerous patches of disease are found scattered throughout the cord, and indeed throughout the nervous system generally, differ from those just described, in that their commencement is more irregular. Thus the disease may begin with paralysis of an arm or paralysis of cranial nerves. The paralysis also spreads in an irregular fashion. Nevertheless, paraplegia is likely to become a prominent feature. Optic neuritis has been observed in this form of myelitis. The result is generally fatal. Further, there is a form of paralysis, the morbid anatomy of which is not fully known, but which appears to depend upon an acute non-progressive disseminated encephalo-myelitis. It commences during the acute stage of an infectious fever, such as smallpox, typhoid, measles, the first symptoms being loss of power of speech, and loss of power in the limbs. But these, owing to the grave constitutional condition of the patient, may go unnoticed for the time. After recovery from the fever the paralysis slowly improves, till at length the patient is left with an ataxia of the limbs, of greater or less severity, associated probably with an increase of the tendon reactions, and impairment of speech due to peculiar articulatory defects. This affection was originally described under the title of "acute ataxia."

DISSEMINATED SCLEROSIS

Disseminated or insular sclerosis is characterised anatomically by the presence of patches of degeneration of irregular size, shape, and distribution, which may affect any part of the central nervous system or even the peripheral nerves. It is, therefore, not a purely spinal disease; but since spinal symptoms are among the most common of its clinical manifestations, it may fitly be considered here.

It is rare in children, commonest in early adult life, and becomes rare again as life advances. Both sexes are subject to it. As to its causation little is positively known. It has been surmised

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that it is due to the toxic effects of acute infectious diseases; clinically this is difficult of proof, though some patients trace the origin of their troubles to influenza or other acute illness. In some other cases depressing mental influences, sudden or persistent, seem to have determined the onset. Frequently no cause can be assigned. It is not, except in very rare instances, a "family" disease (see p. 164).

When the **symptoms** have reached a certain degree of completeness, and when they coincide with the description usually accepted as typical, the disease is easy to recognise. The patient then has weakness in the lower limbs, this paraplegia being of the spastic type; the sphincters may or may not be affected. Definite anæsthesia is not the rule. The upper limbs, and often the head and trunk, exhibit a peculiar "tremor," the main characteristics of which are, that the limb is quiet while not in use, but, on voluntary effort being made, to and fro movements begin, which are irregular both in rhythm and amplitude, and become more marked the greater the precision of movement required—the so-called intention or volitional tremors. Thus, in lifting a cup to the lips, the hand begins to shake as the patient extends it, the oscillations become larger as he approaches the cup, and finally, as he attempts to guide it to his lips, the fluid contents are thrown hither and thither. The speech may be affected in a peculiar way; the words and syllables are not slurred or run together, but unduly separated from each other, so that the speech has been described as scanning, staccato, or syllabic. There is nystagmus, that is to say, the eyeballs oscillate when the patient fixes an object or follows it with his eyes from side to side. These oscillations are usually (not invariably) lateral in direction, small in amplitude, and have a tolerably regular rhythm. They may be compared to the limb tremor.

Optic atrophy is of somewhat frequent occurrence. The intensity of it may vary from a mere pallor of the disc to a well-marked gray atrophy; the visual defect is not always proportionate to the visible atrophy, for in some cases there is much loss of vision while the disc is nearly normal (probably in consequence of disease further back than the papilla); in others there is distinct atrophy without much loss of eyesight.

Cases certainly exist in which these symptoms — paraplegia, tremor, scanning speech, nystagmus—have been steadily evolved, and exist in their most typical form. But it is by no means always so; for, firstly, some of them, even at a late period, may be absent

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or ill-developed. The speech affection is far less common than is usually supposed. The tremor, when well marked, is highly typical of the disease, but in its early stages it may consist of a finer, more rhythmical movement, which is scarcely characteristic. Nystagmus, however, is certainly common, and commoner in this than in any other form of nervous disease. Paraplegia is not the only possible affection of gait; there may be ataxia, or a mixture of ataxia with paraplegia, or the swerving reeling walk of cerebellar disease. All these varieties tend indeed to settle down into a spastic paraplegia, and in all of them it is commonest to find exaggeration of the tendon reactions, and the extensor type of plantar reflex. Secondly, the mode in which the disease develops may be very puzzling. As in other chronic nervous diseases there may be prodromata, such as vertigo, transient squint, temporary troubles of micturition, or numbness and paræsthesia of various kinds. But still more formidable paralyses may occur and get well. The patient may lose the sight of an eye and recover, or may develop a hemiplegia or paraplegia, or paralysis in one limb, which passes off. Indeed there may be a whole train of such recoverable paralyses, spread over a term of several years, in the intervals of which he is well enough to go about, and, except under skilled examination, exhibits no symptoms of consequence.

Diagnosis.—It should be recognised that a history of this kind points to disseminated sclerosis, though we may be unable definitely to diagnose it. But the suspicion of *hysteria* is sure to arise, especially when the patient is a young woman whose manner and general appearance suggest an emotional temperament. Careful search should, in this case, be made for any of those symptoms which indicate organic disease, and therefore negative hysteria pure and simple, such as ocular paralysis, nystagmus, persistent ankle-clonus, plantar reflexes of the extensor type, and above all optic atrophy. Even in the absence of these, it may be well to defer the diagnosis, and to await further possible developments (see p. 329).

Of organic nervous diseases, the following are most likely to need differential diagnosis:—

Intra-cranial tumours, particularly cerebellar tumours.—The symptoms are more likely to be hemiplegic in their distribution; optic neuritis or secondary optic atrophy is more likely to be present; whereas the optic atrophy in disseminated sclerosis is primary.

Paraplegia from transverse myelitis.—Although disseminated sclerosis may present itself under the form of a simple paraplegia, it is commoner to find indications of more widely-spread disease,

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e.g. exaggeration of tendon reactions in the arms (as well as legs), ocular symptoms, etc.

Syphilitic nervous lesions of irregular distribution.—Multiplicity and irregularity of lesions are common in syphilis of the nervous system; improvement and relapse may also take place. Nystagmus, tremor, or perhaps affection of speech would point to disseminated sclerosis.

The tremor of *paralysis agitans* differs as a rule widely from that of disseminated sclerosis, in that it is a rhythmical and less ample vibration, checked rather than increased by voluntary movement. But in some cases of paralysis agitans the tremor is not stopped by movement, and in early cases of disseminated sclerosis the tremor may be fine and rhythmical; so that the diagnosis in special instances may not be easy. Evidence of paralysis agitans is then to be found in the peculiar aspect of the face and the pose of the body, and the gradual development of the symptoms; evidence of disseminated sclerosis in exaggeration of the tendon reactions, nystagmus, and the history of fluctuating and evanescent paralysis.

The tremor of *general paralysis* is much finer than that of disseminated sclerosis, being indeed a tremulousness rather than a vibration of the limbs; the tongue and lips too are tremulous, the speech hesitating and slurred, not syllabic, and there are mental symptoms.

Of the cases classed clinically as *ataxic paraplegia* probably not a few are really disseminated sclerosis; for in this disease there may be a gait which is partly spastic, partly ataxic. When ataxic paraplegia is due to combined system disease of the posterior and lateral columns, it is probable that there will be more definite anæsthesia than in disseminated sclerosis, a more steady evolution of symptoms, with a regular spread of them from the lower to the upper limbs, and also absence of tremor and of nystagmus.

Tabes usually exhibits several points of contrast, as follows:—

In Tabes	In Disseminated Sclerosis
Lightning pains are common	Rare
Knee-jerks are absent	Increased
Pupils do not act to light	React normally
Volitional tremors absent	Usually present
Nystagmus absent	Common
Gait ataxic	Usually paraplegia

The ultimate prognosis is undoubtedly bad. It is likely that

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the patient will eventually become a confirmed paralytic, and die either from intercurrent disease, cystitis, or bed-sore. Yet we must not be too hasty in such predictions. It is only recently that the phase of recovering and relapsing paralysis has been recognised, and we do not accurately know how long this stage may last, nor whether some cases may not stop short in this stage. We think, however, that when the typical form of the disease has developed, with marked and persistent paraplegia, the chances are against even a temporary recovery. But probably most mistakes in prognosis are made in the other direction; symptoms really indicating disseminated sclerosis are regarded as "only hysterical," and a favourable prognosis is given, which the final result discredits.

We know of no cure for this disease; yet **treatment** is not always useless. Many cases improve after admission to hospital, doubtless from the combination of rest, proper food, and nursing. It is likely that many cases have been treated (*quâ* hysteria) by massage and over-feeding, and have received benefit therefrom. Where there is much spasticity, prolonged rest in bed is advisable. Iron, quinine, nux vomica, iodides, hypophosphites, arsenic, cod-liver oil, may be tried and sometimes appear to do good.

SYRINGOMYELIA

The symptoms of syringomyelia are thought to depend upon the fact that there exists in this disease a slowly progressive lesion, commencing in the central region of the cord, and generally at the level of the cervico-dorsal segments, which gradually extends itself both towards the periphery and up and down the cord, giving rise also, it may be, to secondary ascending and descending degenerations (see p. 166).

These **symptoms** are mainly as follows:—

"Dissociated" anæsthesia.—Heat and cold cannot be distinguished; generally, too, there is anæsthesia to painful stimuli; yet sensation to touch is normal. Usually, this peculiar anæsthesia is found in the upper limbs and upper part of trunk and neck; but it may spread, and indeed its distribution from the first depends on the localisation of the mischief in the cord. Later on, sensation to touch may also be impaired.

Muscular atrophy, commencing commonly in the hand muscles, and spreading very slowly to fore-arms, arms, and trunk. It doubtless indicates that the anterior cornua are involved, and

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indeed is indistinguishable, *per se*, from the atrophy of chronic anterior poliomyelitis.

Trophic symptoms, other than muscular atrophy, may occur, mostly in the upper limbs. Such are disease of the joints, very like the joint disease of tabes, presently to be described; necrosis of bones, by which the fingers may be destroyed or deformed; painless whitlows; bullous and other affections of the skin.

Spinal deformity is common, either lateral or antero-posterior curvature, or a combination of the two.

Paralysis of the cervical sympathetic may be found, due, no doubt, to disease of the upper dorsal segments whence these nerves originate.

As the disease slowly develops, other symptoms may follow, due either to spread of the original lesion, or to secondary degenerations. Thus the lower limbs become affected either with a paraplegia of the spastic type, or in some instances with ataxia, anæsthesia, and absence of tendon reactions. Upward spread of the disease may affect the origins and central connections of the cranial nerves, and be indicated by laryngeal paralysis, difficulties of swallowing, nystagmus, or reflex iridoplegia.

The course of the disease is very chronic. It may progress slowly for years, or such slow progress may be interrupted either by periods of quiescence, or by somewhat sudden accessions of new paralysis. These sudden developments have been ascribed to hæmorrhages taking place in the diseased tissue.

No cure for it is known; we can only endeavour to maintain the general health and treat symptoms as they arise.

MORVAN'S DISEASE, so called after the French physician who described it, is now commonly considered to be a variety of syringomyelia, the chief peculiarities of which are—that the symptoms are mainly confined to the upper extremities, and that trophic disturbances, notably painless whitlows, are a predominant feature.

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B. SYSTEM DISEASES OF THE CORD—i.e. DEGENERATIONS AFFECTING THE SEVERAL SYSTEMS OF NEURONES

DEGENERATION OF THE AFFERENT NEURONES: POSTERIOR SCLEROSIS = *TABES DORSALIS OR LOCOMOTOR ATAXY.*

DEGENERATION OF THE UPPER EFFERENT NEURONES: LATERAL SCLEROSIS = *PRIMARY SPASTIC PARAPLEGIA.*

COMBINED DEGENERATION OF AFFERENT AND EFFERENT NEURONES; POSTERO-LATERAL SCLEROSIS = 1. *"ATAXIC PARAPLEGIA"*; 2. *GENERAL PARALYSIS*; 3. *HEREDITARY ATAXIA*; 4. *SPINAL DEGENERATIONS IN ANÆMIA.*

DEGENERATION OF THE LOWER EFFERENT NEURONES = PROGRESSIVE MUSCULAR ATROPHIES OF SPINAL ORIGIN. 1. *CHRONIC ANTERIOR POLIOMYELITIS* OR *PROGRESSIVE MUSCULAR ATROPHY*, OR 2. COMBINED WITH DEGENERATION OF THE UPPER EFFERENT NEURONE = *AMYOTROPHIC LATERAL SCLEROSIS.*

(See pp. 39, 40, 41.)

TABES DORSALIS OR LOCOMOTOR ATAXY

The **morbid anatomy** of tabes consists mainly in chronic degeneration of the posterior nerve roots, and of those parts of the posterior columns which form their continuation. This usually begins in the lower levels of the cord (sacral, lumbar, or lower dorsal segments), and gradually attacks the nerve roots at higher levels. In exceptional cases (cervical tabes) it may begin in the upper cord. In old cases the disease is appreciable by the naked eye; the posterior nerve roots are thinned; the posterior columns are firm in consistence, gray in colour, and shrunken; the pia covering them may be thickened. In early stages the disease can be detected and more accurately localised in suitably-stained microscopic sections. Certain groups of fibres are affected earlier than others, notably perhaps Lissauer's tracts (fine fibres which run up the cord along the tip of the posterior cornu), and fibres in the outer part of Burdach's columns (described by Charcot as "bandelettes externes"), but before long all the fibres entering from the posterior roots suffer. As some of these run without break *viâ* the columns of Goll to the medulla, the net result is—in the upper

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levels of the cord, degeneration of Goll's columns only; in the lower parts of the cord, degeneration of the whole of the posterior columns (except a few so-called "endogenous" fibres which we need not here specify). It may happen that the cells of Clarke's column, and the ascending cerebellar tracts which spring from them, degenerate also; but this is not invariable. Tabes then is a "system" disease, the system of fibres affected being those which enter by the posterior nerve roots. As these are under the nutritive sway of the posterior root ganglia, disease of these ganglia has been looked for, and has been found, it would appear, in some cases but not in all (see Plate IV. Fig. 1, facing p. 41, and p. 162).

Such is the essential anatomy of tabes; still, the disease may have a wider incidence. Atrophy of the optic nerves is not uncommon; degeneration of peripheral nerves may occur; and lastly structures altogether outside the sensory sphere may degenerate, viz. the motor nuclei in the pons or medulla.

Etiology.—Tabes usually commences in middle life; it is much more common in men than in women; and most authorities consider that it has some connection with past syphilis, a view which is based on statistics which show that an overwhelming proportion of the patients have had syphilis. What the connection is still remains uncertain; but a plausible view is that syphilis, like other more acute infectious diseases, produces in the body a toxin which may have a special affinity for nerve tissues and even for a certain system of nerve fibres. Thus the afferent nerve system of the cord breaks down; and sometimes, as we have indicated, other nervous structures too.

The syphilitic theory must not, however, be made too absolute. In a few cases syphilis cannot reasonably be presupposed; perhaps in these some other toxin may have been at work. Further the very marked preponderance of male patients over female cannot be sufficiently explained by syphilis. Neither can we, accepting syphilis as the prime predisposing cause, ignore the action of exciting causes, such as exposure to cold, fatigue, injury, sexual excess. These it would seem may determine the onset, or may light up the disease when latent.

Tabes is not hereditary; we doubt whether even a "general nervous heredity" really contributes to it.

Symptoms.—The disease is commonly divided into three stages—a preliminary stage, in which the movements of the patient are not materially embarrassed; an "ataxic" stage, characterised by inco-ordination of muscular action; and a "paralytic" stage, in

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which walking and standing become impossible. The duration of these stages may vary very greatly; indeed the disease may never pass on from the one into the next.

In the *first stage* tabes is recognised by the concurrence of three symptoms: (1) Pains, usually of the kind known as "lightning pains," severe momentary twinges, occurring in bouts with free intervals, not limited to any one nerve district, generally but not invariably commencing in the lower limbs. They are often put down to "neuralgia" or "rheumatism." Another less characteristic pain is a more continuous aching or boring pain in the back or limbs. (2) Loss of knee-jerk. The knee-jerks are absent, as a rule, when the patient first seeks advice; but sometimes they may be caught in the course of disappearance, and are then feeble, uncertain, perhaps unequal on the two sides. Since their absence is due to sclerosis of the posterior roots at the lumbar level, they may be preserved in rare cases (cervical tabes) when the disease begins at a higher level. (3) Reflex iridoplegia. This phenomenon, otherwise known as the "Argyll-Robertson pupil," consists in the fact that the pupil does not contract when light is thrown on the retina, whereas it contracts normally during the effort of convergence and accommodation. Sometimes, though this is less characteristic, it contracts neither to light nor convergence. Often too the pupils are very small (miosis). The diagnostic importance of reflex iridoplegia is due to the fact that it is very common in tabes, rare in other nervous diseases, except general paralysis. Some few cases of tabes do not present this sign.

Although the preliminary stage of tabes may manifest itself by no other symptom than these three, yet in it we may be confronted with certain puzzling and varied symptoms which we shall presently mention. Some symptoms, however, occur so frequently as to deserve mention now, these are (α) transient diplopia, (β) troubles with micturition, either in the way of retention or incomplete power of control. These may help to suggest, or to corroborate, a diagnosis of early tabes.

With the *second stage* comes the muscular inco-ordination, which has given rise to the well-known name "locomotor ataxy." This usually begins in the lower limbs. The gait becomes unsteady, particularly when the patient essays to turn quickly, to go up and down stairs, or to walk in the dark. Perhaps before this he may have noticed that he is easily fatigued by walking, or that his legs sometimes give way suddenly. As the unsteadiness gets worse, we see that he plants his feet wide apart, and looks at them as he

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walks. Finally (and often not till quite a late period in the disease) he develops the typical "ataxic gait." He walks slowly, generally with sticks, leaning forward and watching his feet, and at each step lifts the advancing foot abruptly, too high, and too far out, and then brings it down heel first with a stamp.

"Romberg's symptom" is a fairly delicate test of ataxy. The patient should be made to stand with his feet close together, without support, and then to close his eyes. He forthwith begins to sway and to fall. In time the ataxy involves the upper limbs, when it reveals itself as an awkwardness in fine movements of the hand and fingers, or for actions such as adjusting the back of the dress, where the eyesight cannot aid. In picking up a pin from the table the hand hovers over it before it can grasp it. If the patient be told to close his eyes, and touch the tip of the nose quickly with his forefinger, he goes wide of the mark.

Along with the ataxia two other conditions are often found, each of which has been supposed to stand in some causal relation to it. The one is anæsthesia. Anæsthesia of some form or other almost always accompanies severe ataxia. It is usually most marked in the legs. Any or all of the modes of sensation (tactile, thermal, painful, muscular) may be involved. Delay in the transmission of painful stimuli is frequent. In the upper limbs sensory affections are apt to begin on the ulnar side of hand and forearm; analgesia of the ulnar nerve, when roughly pressed upon, has been described as an early symptom. The other condition has been called "hypotonia" of the muscles. This means that the tonic contraction which muscles in their normal state exhibit is lessened. The fact is shown by the ease with which extreme passive movements of the joints may be carried out. Thus it may be possible, having laid the patient flat on a couch, to flex the thigh almost completely on the abdomen; or, flexing the thigh and keeping the knee extended, to bring the limb to an acute angle with the body; or, after semiflexing the knees and hips, to abduct the thighs till they are almost flat on the couch; or for the patient sitting up in bed with the knees extended to put the head right down between the knees. Normally these movements are resisted by the tonus of those muscles which are the opponents of the several movements described.

In the *third or "paralytic" stage* there may be real paralysis. Indeed, even earlier than this, not a few cases exhibit some muscular weakness in addition to muscular inco-ordination. But usually this stage may be taken to mean that ataxy has advanced

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so far that the patient cannot stand or walk. Probably other symptoms will have advanced too: anæsthesia will be more extensive and complete, and the functions of the bladder more permanently damaged. Hence danger to life becomes more imminent from cystitis, from bed-sore, or from intercurrent disease.

The uniformity of the disease is in practice often broken by the appearance of striking symptoms which may involve very diverse functions and parts of the body, and which have little apparent connection with the main disease. And since many of these are apt to occur in the early stages of it, their true meaning is often not appreciated. Among such are the following:—

Optic atrophy.—This is fairly frequent, and may be the first symptom. It is primary (*i.e.* not preceded by optic neuritis), causes limitation of the fields and progressive loss of central vision, attacks either both eyes at once, or one after the other, and usually results in complete blindness. It is said that cases which begin in this way develop ataxy of the limbs either very slowly or not at all.

Ocular paralyses.—These are common as early symptoms; they are often quite transient, but sometimes remain unaltered for long periods. They may vary from a mere diplopia to a marked squint or ptosis. Often the paralysis is confined to a single nerve district, third, sixth, or fourth. A rarer form is an ophthalmoplegia (probably due to nuclear degeneration), which is progressive and gradually involves all ocular movements.

Other motor paralyses. — Transitory hemiplegia or paraplegia may appear in the early stages. Hemiplegia due to organic causes (hæmorrhage, thrombosis, etc.) may occur as an accidental complication. Paralysis sometimes arises in the district of certain nerve trunks, *e.g.* the hypoglossal (causing hemiatrophy of the tongue), the spinal accessory, or the peroneal nerve. Progressive muscular atrophy is an occasional but very rare event.

Visceral "crises."—These are sudden paroxysmal disturbances of function, often recurrent, sometimes periodical, and generally associated with pains. The best known are the gastric crises. In these, either without warning or during an attack of lightning pains in the back or epigastrium, vomiting sets in and may persist for several days in a most severe and intractable form. For its onset and for its cessation there is usually no assignable cause. Similarly paroxysmal attacks of diarrhœa, of tenesmus and other painful sensations in the rectum, have been called intestinal or rectal crises. The "laryngeal crisis" in its worst form is a very sudden and severe dyspnœa, even accompanied by loss of consciousness. This

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is very rare; more commonly paroxysms of dyspnœa in tabetic patients are due to the temporary aggravation of a curious laryngeal paralysis from which such patients sometimes suffer, viz. paralysis of the muscles which abduct the vocal cords.

Reflex abnormalities.—We have spoken of the tendon reactions. Of skin reflexes we have little to say; they will probably be absent when there is cutaneous anæsthesia. As to organic reflexes, those governed by the lower part of the cord may be affected at an early period. "Precipitate micturition" (the patient having to hurry to pass water directly the desire takes him) is then very common; sometimes this becomes actual incontinence. Retention may also occur. Sexual capacity (or at least the capacity for abnormally frequent repetition of the sexual act) is, according to some authors, increased in the early stages of tabes. But impotence may be an early phenomenon, and is certainly common later on.

Trophic changes.—The most striking of these is joint disease (tabetic arthropathy, Charcot's disease of joints). A joint, usually the hip or knee, which has been the site of lightning pains, of some slight crepitation, or of some trivial injury, or concerning which there has been no complaint whatever, becomes suddenly and greatly swollen. There may be fluid not only in the joint, but in the tissues of the limb around. Strange to say there is no pain, as in ordinary acute synovitis. In "benign" cases this swelling may pass away and leave the joint but little injured. But more commonly, as the swelling subsides, it becomes evident that the joint is permanently damaged, either by absorption of ligaments, cartilages, and bone, which leaves an extreme and "flail-like" mobility; or by the throwing out of buttresses and bosses of osseous material which limit the proper movements, or by a combination of these processes. Closely associated with the joint disease, though less frequently in evidence clinically, is disease of the bones themselves. They may become rarified, so that spontaneous fractures result. Combined disease of the tarsal joints and tarsal bones may cause a peculiar deformity of the foot. Perforating ulcer of the foot is not uncommon. It commences, as a bad corn, usually in an anæsthetic area, and beneath this a suppurating sinus forms. It may get well, or may become so troublesome as to require amputation. Other possible trophic changes, such as spontaneous rupture of tendons, falling out of teeth, herpes, subcutaneous ecchymosis, need only be mentioned.

Mental symptoms.—The mind is usually clear throughout the disease, but sometimes excitement, exaltation, and delusions re-

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calling those of general paralysis of the insane set in. Recovery from this condition is not impossible, but, on the other hand, the patient may pass over into the hopeless condition of a general paralytic. Sometimes it may be difficult from the beginning to say whether the case should be classed as tabes or as general paralysis; indeed the two diseases are so closely allied, anatomically and etiologically, that it is surprising clinical distinctions do not oftener fail us.

With respect to **diagnosis**, advanced cases of tabes present little difficulty. Nor indeed do early cases, when the three cardinal symptoms, lightning pains, absence of knee-jerk, and reflex iridoplegia, are all present. Difficulty may arise when only one or two of these symptoms can be found; or again when some accessory symptom, such as vomiting or joint disease, so occupies our attention that we forget to look for the others. The diseases which will most likely give rise to confusion are—

Peripheral neuritis.—In this the pupil is normal; the disease develops more rapidly; there is usually some closely antecedent factor, such as alcohol, diphtheria, or diabetes.

General paralysis.—Lightning pains are less common; the onset is generally more rapid; and careful examination will probably disclose some indication of mental degeneration, affection of speech, or tremor of tongue. But there will remain cases which it is very difficult to classify.

Intracranial disease.—In some cases of long-standing cerebral or cerebellar tumour there may be optic atrophy, absence of knee-jerk, paroxysmal vomitings, unsteadiness of gait, which may to a certain extent simulate tabes.

Prognosis.—Tabes is an incurable disease; but its progress is usually slow, indeed may be measured by years; and some cases appear to come to a standstill. Much less commonly the ataxia comes on quickly—in a few months, or even weeks. We think that when ataxia has developed steadily and become thoroughly established it is likely to be permanent. On the other hand, some of the most distressing symptoms, such as the pains and the vomitings, may pass off entirely.

Treatment.—All patients of this class should be enjoined to live quietly and simply; all sources of nervous exhaustion, physical or mental, should be avoided; the utmost moderation in alcohol, tobacco, and, above all, in sexual relations, must be observed. Adverse climatic conditions promote the pains; and therefore a warm, equable climate and a dry subsoil should be sought after, if

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possible. For the general condition the following drugs have been recommended: nitrate of silver, iodide of potassium, arsenic, belladonna, chloride of aluminium. Some authors advise a mercurial course, others discountenance it. Hydropathic treatment and electrical treatment may also be tried. For the pains it is best first to give such drugs as phenacetin, antipyrin, or antifebrin, but in some cases morphia cannot be avoided. The bladder may often require careful surgical treatment. For the inco-ordination, the method of graduated exercises seems to be the most promising mode of treatment. The patient, unable, it may be, to stand or walk alone, is made to practise, lying down, movements of the legs, at first of the very simplest kind, then others requiring more power of direction, then more complicated ones, the greatest care being taken to avoid over-fatigue; till at last he may be brought to standing and walking. In less severe cases, a certain amount of moderate daily exercise is advisable, always short of fatigue.

SPASTIC PARAPLEGIA

Just as tabes is a degeneration of the afferent neurones, mainly of those in the posterior columns, so we might expect that there should exist a primary degeneration of the efferent neurons, that is to say of the pyramidal tracts, which run principally in the lateral columns. And such a disease has been described under the title of "lateral sclerosis."

The **symptoms** would be weakness, with increase of tendon reactions, beginning in the lower limbs, the development of a spastic gait, followed by a complete paraplegia with rigidity, and upon this, as time goes on, would supervene similar phenomena in the upper limbs. There must be no affection of sensation, no paralysis of the sphincters, no atrophy of muscle, for any one of these symptoms would indicate the implication of other parts of the cord over and above the pyramidal tracts.

Without denying that such cases may exist, it may be safely said that a primary progressive uncomplicated lateral sclerosis is a very rare disease. Many of the cases so-called will be found on careful examination to be really the sequelæ of an incomplete transverse myelitis, and therefore neither primary nor progressive. Others may turn out to be amyotrophic lateral sclerosis, disseminated sclerosis, cerebral diplegia, or even due to hysteria. Perhaps the best instances are those in which a spastic paralysis, affecting the legs, the arms, and lastly the functions of speech, has appeared in

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several members of a family. But this hereditary spastic paraplegia is extremely rare, and moreover in the single recorded post-mortem some of the ascending tracts were degenerated as well as the pyramidal tracts. Still the clinical phenomena corresponded to those of an idiopathic lateral sclerosis.

ATAXIC PARAPLEGIA AND OTHER FORMS OF COMBINED SYSTEM DISEASE

The title of "ataxic paraplegia" has been given to a certain grouping of symptoms which appear to indicate a degeneration proceeding in the lateral and posterior columns of the cord simultaneously (see p. 162).

Symptoms.—In this condition the phenomena of spastic paraplegia and of ataxia will be combined, the one or the other predominating according as the lateral or the posterior columns are the most affected. In the bulk of such cases the paraplegia predominates, that is to say, the legs are weak, the gait dragging, and the tendon reactions exaggerated. But along with this there may be unsteadiness, which is increased on closing the eyes, and uncertainty and irregularity in the movement of the feet; and as the disease becomes more extensive in the posterior columns the knee-jerks may disappear, though the paraplegia remains. Difficulties with micturition, like those observed in tabes, may occur, and possibly ocular and papillary phenomena suggestive of that disease. It is said that there may be nystagmus. Lightning pains and anæsthesia may be present, but perhaps are less frequent than in ordinary tabes. The gradual evolution of such an ataxic paraplegia, involving first the legs and then the arms, may perhaps be taken as sufficiently indicating a combined degeneration of the afferent tracts in the posterior columns and of the pyramidal (efferent) tracts. But certain considerations should be remembered which go to qualify this statement. First, that the symptoms of ataxic paraplegia may be produced by disseminated sclerosis; indeed we think it probable that many cases are really of this nature, and not due to combined tract degeneration. Secondly, that where such a combined degeneration really exists, the one set of symptoms may during life throw the other set quite into the shade. Thus a case which during life appeared to be little more than a progressive spastic paraplegia may exhibit, post-mortem, changes in the posterior as well as in the lateral columns; and reversely a case which during life corresponded fairly well with tabes may exhibit lateral as well

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as posterior sclerosis. Thirdly, that combined tract degeneration may, and indeed does, frequently occur under conditions which themselves must be ranked as distinct clinical entities, the spinal degeneration being more or less subsidiary. These will now be briefly described.

General Paralysis of the Insane

The commonest and most important is general paralysis of the insane. In its best known and most striking form this disease is mainly cerebral, and as such it has been already described (p. 99). But spinal degenerations, sometimes of the pyramidal tracts, sometimes of the posterior columns, or of both together, often co-exist with the cerebral disease.

Hereditary Ataxia or Friedreich's Disease

Another disease characterised anatomically by combined degeneration of the posterior and lateral columns is that known as "hereditary ataxia" or "Friedreich's disease." The few cases that have been examined post-mortem have been of long standing, and in these it has been found that the posterior columns were in an advanced stage of sclerosis, and the lateral columns (pyramidal tracts and sometimes the ascending cerebellar tracts) in a less advanced stage. The chief **clinical features** are, firstly, that the disease runs in families, so that several members of a generation, brothers and sisters, or perhaps cousins, are attacked. Sometimes, though less commonly, it appears in successive generations. Secondly, as to the individual symptoms; the principal feature is a slowly progressive ataxia, affecting the lower limbs first and afterwards the upper limbs. The tendon reactions are in the large majority of cases abolished. So far there is an evident resemblance to tabes, but there are many differences. The onset is at an early age, most frequently perhaps about the time of puberty, sometimes in childhood, always before twenty-five. Moreover, as we have already said, it is a "family" disease, which tabes is not. There is an absence, at any rate in the early stages, of lightning pains and of anæsthesia, which are the rule in tabes, and, generally speaking, of the accessory symptoms of tabes, such as optic atrophy, bladder troubles, visceral crises, trophic disturbances. The pupil too reacts normally to light and accommodation. On the other hand, Friedreich's disease presents certain features of its own.

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As the disease advances the speech is affected, becoming elisive, slurring, and indistinct. At a later stage nystagmus may develop. Curvature of the spine, scoliosis or kypho-scoliosis, is often observed, and may be an early symptom. A peculiar deformity of the feet, pes cavus, with drawing up of the toes, often develops, and as time goes on the lower limbs may become powerless and more or less contracted, symptoms which may probably be referred to implication of the lateral columns. Chorea, or a condition indistinguishable from it, frequently figures in the early history of these cases; and choreic movements of the face may remain permanently. There is often too a general unsteadiness and shakiness of the head and trunk. These symptoms, coupled with the expressionless aspect of the face, the indistinct speech, and the awkwardness of the limbs, suggest to the observer that the patient is half-witted; but the mental state is generally fair, allowance being made for the disadvantages entailed by a chronic paralysis. The disease is slowly but surely progressive; nothing can be done for it, save to keep the general health at the best possible level, and to shield the patient, so far as possible, from acute diseases, for these may have a most deleterious after-effect upon his paralysis (see p. 163).

HEREDITARY ATAXIA, "CEREBELLAR" FORM.—A rare form of "hereditary ataxia" has been described, which, like Friedreich's disease, runs in families, and first produces disabilities in walking, afterwards affects the upper limbs and organs of speech. It differs in the following particulars: The age of onset is later, commonly after twenty; the knee-jerks are preserved or exaggerated, there may be even ankle clonus; optic atrophy is frequent; paresis of ocular muscles, and a peculiar kind of ptosis may be present. The speech has a jerky, explosive character, and irregular movements of the limbs and facial muscles appear to be more marked than in Friedreich's disease. It is thought by some authorities to be due to chronic progressive atrophy of the cerebellum, and has therefore been called hereditary cerebellar ataxia.

SPINAL DEGENERATION IN CONNECTION WITH ANÆMIA

The third condition is grave anæmia. By this expression we mean to exclude the bulk, at any rate, of cases of chlorosis, and to include the severer forms of anæmia, whether they are strictly to be called "pernicious" or not. It is now established that spinal degeneration may arise in connection with such anæmia. What the exact connection is remains somewhat doubtful; but from the

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fact that sometimes the anæmia appears first, and sometimes the spinal symptoms, it seems probable that both are dependent on some common cause. Possibly this may prove to be the presence of a toxin in the system. Into the details of the morbid anatomy we need not enter, save to say that degeneration is found in both the posterior and the lateral columns, sufficiently symmetrical and regular to be regarded as a "system degeneration." In some cases scattered foci of disease have been seen, which appear to be the remains of hæmorrhages, analogous to the hæmorrhages which are known, in severe anæmia, to occur in the retina. But that the degeneration is not secondary to these is shown, partly by its symmetry and regularity, partly by the fact that often no such foci can be found. The symptoms are those of "ataxic paraplegia," that is, weakness with tendency to rigidity on the one hand, ataxia with sensory and bladder symptoms upon the other, combined in variable proportion. But the disease runs a comparatively rapid course, many cases terminating fatally within two years or less.

There appears to be a certain group of cases to which a very definite march can be assigned. Some of these, it should be observed, occur in connection with anæmia and some do not. The first stage is that of a mild ataxic paraplegia. There are paræsthesiæ such as numbness, stiffness, and tinglings, mainly in the lower limbs. The gait is clumsy and somewhat dragging, the tendon reactions are exaggerated, the plantar reflex is of the extensor type. In the second stage (and it should be noted that the changes of phase may occur suddenly) the patient loses all power of walking. This appears to be mainly due to a great increase in the sensory paralysis, especially paralysis of the sense of position, although the motor paralysis also now begins to get worse. The spasticity of the limbs continues, or increases. There may be pains of various kinds—lightning pains in the limbs, girdle pain, dragging pain around the lower chest; sometimes there is slight incontinence of urine. Irregular pyrexia may occur at this or indeed at any other stage of the disease. In the third stage the motor paraplegia becomes absolute. The rigidity gives way to flaccid paralysis; the knee-jerks disappear. The muscles waste. Œdema of the limbs and loins appears. There is complete incontinence of urine and fæces. The general condition changes for the worse, fever, delirium, or extreme prostration sets in, and death follows. Post-mortem, a system degeneration has been found, of the afferent and efferent neurones in the posterior and lateral columns, and, in addition, a widely spread destruction of the white

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matter at the mid-dorsal level, leaving intact only the gray matter and a narrow border of white matter around it.

PROGRESSIVE MUSCULAR ATROPHIES OF SPINAL ORIGIN

We now turn to the diseases characterised—clinically, by the symptom of progressive muscular atrophy — anatomically, by degeneration of the lower efferent neurones, that is to say of the anterior cornual cells, the anterior nerve roots, motor fibres of the peripheral nerve trunks and branches, and of the muscles, one or all of these constituents being involved (see p. 163).

Amyotrophic Lateral Sclerosis

But the first disease of this class which we shall mention, namely amyotrophic lateral sclerosis, has this peculiarity that both upper and lower neurones are involved, sometimes indeed in their whole length; and thus, just as tabes is a systematic degeneration of the afferent nervous apparatus, so is this disease of the efferent.

In all cases of amyotrophic lateral sclerosis there is found—on the one hand, degeneration of the anterior cornual cells, and secondarily to this (perhaps in some cases also primarily) degeneration of the fibres which pass from these cells to the muscles, and of the muscles themselves; on the other hand, degeneration of the pyramidal tracts in the cord. This degeneration of the pyramidal tracts has in some cases been traced up as far as the medulla and pons; in some few other cases up through the corona radiata to the cortex cerebri, wherein the cells that stand at the head of the motor tract have been found to be diseased. Thus the whole motor system may be involved. It was at one time held that the disease begins in the pyramidal tracts, and is propagated thence by a sort of physiological continuity to the anterior cornual cells. It seems probable, however, that the disease may strike at either upper or motor neurone independently or simultaneously.

The **etiology** of the disease is practically unknown. It does not run in families; it has no syphilitic basis, so far as we know. Exposure, fatigue, and nervous strain of various kinds sometimes figure in the history. Both sexes may be attacked. It occurs mainly in early adult and adult life (æt. twenty-five to fifty).

Clinically it presents the curious combination of atrophic and spastic paralysis.

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In its best-known form the disease may be described as follows:—The first symptom is often pain, or paræsthesia, such as numbness or tingling in the arms and hands. But sensory symptoms never go farther than this, and there is no real anæsthesia from first to last. Weakness and wasting in the upper limbs are next noticed; the weakness, it is affirmed, is greater than would be expected from the amount of wasting. The atrophy, which at first may not be considerable, but which tends to make progress rapidly, usually begins in the intrinsic muscles of the hands; the muscles of the forearms soon get thin; and the upper arms, notably the deltoids, suffer before long. But on the whole there is less picking out of individual muscles than in the more chronic form of spinal amyotrophy. Spontaneous twitchings (fibrillary and fascicular tremors) are generally to be seen in those muscles to which the disease is spreading. The electro-contractility of the muscles diminishes as they waste, and usually under the galvanic current there can be found alterations of the polar formula and sluggishness of contraction. Later, all electro-contractility may disappear. Although one upper limb may be attacked before the other, both are before long involved. Even in the presence of this atrophy evidence of the underlying contraction may be obtained; firstly and principally, by an excess of tendon reaction at wrists and elbow, demonstrable till the muscular wasting has become extreme; secondly, by the position into which the hands and fingers are sometimes drawn by contracture of the less paralysed muscles; thirdly, by a peculiar slight resistance which the limb shows under manipulation, called by Charcot "flexibilitas cerea."

The *second* stage is marked by a spastic paralysis of the legs, which may indeed be succeeded later by atrophy. From the first the tendon reactions will have been exaggerated, in both lower and upper limbs, and even in parts above, such as the jaw. The paraplegia is not accompanied by any affection of the sphincters, nor by any anæsthesia, and herein it differs from the paraplegia of transverse myelitis.

In its *third* stage the disease involves the motor nuclei of the medulla, and particularly the hypoglossal. The lips become affected; labials are badly pronounced; the patient cannot whistle; the lower part of the face assumes a vacant or lachrymose appearance. (Quite apart from this appearance the patient may exhibit an emotional proclivity, being easily moved to tears or laughter, though it does not appear that either really represent his feelings.)

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The tongue shows fibrillary tremors, and then becomes atrophied. The palate may be paralysed. Speech becomes nasal, and more and more unintelligible; swallowing is difficult and death may easily occur either from the passage of food into the air passages or the extension of the disease to the cardiac or respiratory centres.

This typical form of amyotrophic lateral sclerosis has a rapid course, generally proving fatal within some two or three years.

But all cases do not correspond to the above type; for variations in the point of first incidence of the degeneration may introduce considerable variations into the clinical picture. Thus there are doubtless cases in which the pyramidal tracts suffer first, and paralysis with rigidity is for a long time the only feature, muscular atrophy not supervening till a late period. In others, again, rigidity is quite in the background, partly, it may be, because the pyramidal tracts are not deeply affected, partly because the motor cells for the lower as well as for the upper limbs are affected early, and thus muscular atrophy predominates from the first. Again, in a certain group of cases the nuclear disease begins in the medulla. Bulbar paralysis is then the first symptom, not the latest; and atrophy of the limbs begins afterwards. Here too there is not commonly a well-defined rigidity of the limbs; increase of tendon reactions and perhaps ankle clonus may be the only indication of the pyramidal tract disease. The "jaw-jerk" is exaggerated, and there may be "jaw clonus."

The **diagnosis** has to be made from cervical paraplegia due to various causes, such as pachymeningitis, caries, tumour. In these conditions we may meet with the combination of atrophic paralysis in the upper limbs, and spastic paralysis of the lower limbs. But the atrophy is accompanied by anæsthesia in the upper limbs, and as the cord becomes more deeply involved there will appear anæsthesia of the trunk and legs and sphincter troubles. These, as we have said, are absent in amyotrophic lateral sclerosis. Syringomyelia may present a similar combination, but here again the peculiar sensory affection will guide us, and the evolution of the disease is much more gradual. From the other forms of progressive muscular atrophy amyotrophic lateral sclerosis is distinguished by the spastic symptoms, by the fact that the atrophy more often involves whole segments of the limbs and has less tendency to pick out individual muscles, and by its more rapid course.

The **prognosis** is very bad. No doubt some cases progress less rapidly than others, and perhaps looking to the analogy of

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other degenerations, it would be going too far to say that the disease can never come to a standstill; still we cannot reasonably expect this. Among the remedies usually employed are electricity (the galvanic current by preference), massage, injections of strychnine in cases where the atrophy predominates, iodide of potassium, arsenic, cod-liver oil, and such general tonics as seem best suited to the particular case.

Chronic Anterior Poliomyelitis, or Muscular Atrophy of the Aran-Duchenne Type

Another form of progressive muscular atrophy is due to degeneration of the anterior cornual cells, and apparently of these alone. As they perish, the corresponding fibres in the anterior roots and in the nerve trunks also degenerate and the fibres of the muscles waste. But there is no degeneration of the upper neurones, that is of the pyramidal tracts. Such at least is the ordinary view, but some authors maintain that in all cases of spinal amyotrophy there is degeneration of the pyramidal tracts to a greater or less extent. In that case the distinction between this disease and amyotrophic lateral sclerosis would be one only of degree.

We know little of the **etiology** of this affection, save that it occurs mainly in adults, and does not generally run in families (with the exception to be presently made). Over-use of the limb first affected; exposure to cold, injury, anxieties — such are the causes that patients may adduce. Metallic poisons, or toxins resulting from disease, may perhaps be added, but this is not proven.

Clinically this form begins by weakness and wasting of muscles, and most commonly of the small muscles of the hand. The onset and the spread of the disease are more gradual than in the case of amyotrophic lateral sclerosis; there is not the rapid thinning of the muscles of the forearm and upper limb; but groups of muscles are picked out at considerable intervals. This grouping follows, it is said, the arrangement of muscle representation within the various segments of the cord, and thus a distinction may be afforded from non-spinal amyotrophy. There may or may not be fibrillary tremors. The electro-contractility of the muscles, to both faradism and galvanism, fades in proportion as they dwindle in bulk, and eventually may be quite extinguished. Probably some traces of reaction of degeneration (alteration of polar formula or sluggishness of muscle twitch) will exist; but they may be difficult to demon-

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strate: firstly, because this reaction is always less complete in chronic than in acute muscular atrophy; secondly, because healthy muscle fibres may remain by the side of the diseased ones and mask the special reaction. The progress of the disease is slow, extending over years. If not bilateral from the commencement, it becomes so as a rule; though it need not be symmetrical upon the two sides. The intrinsic muscles of the hands (irrespective of their nerve trunk supply), the flexor group of the muscles on the forearm, the deltoid spinati and biceps group, are most likely to suffer. Bulbar paralysis may supervene eventually. But some cases, it would appear, cease to be progressive, and undergo a natural arrest. The prognosis, therefore, while not favourable for recovery, is far more favourable than in amyotrophic lateral sclerosis, either for the chances of life, or even for the preservation of such muscles as are left.

The **diagnosis** from amyotrophic lateral sclerosis turns on the less rapid course, and on the total absence of spastic symptoms. The knee-jerks are not exaggerated and there is no ankle clonus. The points of diagnosis from myopathic muscular atrophy will be mentioned presently. Neuritis of the ulnar or median nerve trunk will produce muscular atrophy in the hand; but such atrophy is limited to the distribution of the particular nerve trunk, and there either is or has been anæsthesia of a like distribution. There are, however, certain cases of muscular atrophy of the hand muscles in which muscles supplied both by the median and ulnar nerves may be involved, and in which sensory symptoms are slight or absent. Such cases it may be impossible at first to differentiate from a chronic anterior poliomyelitis. But there is this important distinction that they may get well: a fact which would indicate, we presume, that they are due to disease of nerve fibres rather than of nerve cells.

The **treatment** for this, as for most other forms of muscular atrophy, consists in electrical applications, massage, and general tonics. Injections of strychnine may be given freely, since the paralysis is purely atrophic, not spastic in character.

A very rare form of muscular atrophy, in which the anterior cornual cells have been found in a state of degeneration, may be briefly noticed here. The peculiarities of it are: that it attacks infants, and that it runs in families. The children, who in several instances have been previously very fat, begin to evince weakness in the muscles of the thighs and back, generally between the ages of six and twelve months. This spreads to the upper limbs, legs, and

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hands; and the muscles pass into a state of atrophic paralysis. It would appear, however, that the disease in its progress does not pick out special groups of muscles, according to their spinal representation. It proves fatal within two or three years.

MUSCULAR ATROPHY OF THE PERONEAL TYPE

Here too may be noticed a type of muscular atrophy, which seems to stand midway between those forms which are essentially due to disease of the nervous apparatus, and those due to disease of the muscles. The pathology of this tyye is not fully determined; some authors hold that it is due to degeneration of the nerve trunks and branches. Clinically it has been termed the "peroneal" or "leg" type of atrophy, from the fact that it begins in the muscles of the leg. In childhood the feet begin to turn in, or a pes cavus is produced, for which tenotomy is often performed. The legs, and after them the thighs, then begin to waste. A peculiar fact is sometimes noticeable about this wasting, that it may affect principally the lower part of the leg or of the thigh, so as to produce a conical or spindle appearance in this segment of the limb. Afterwards, it may be many years afterwards, wasting begins in the hands, and slowly involves the upper limbs. This peroneal atrophy has often been observed to affect several members of one family; in some instances it seems to have followed some acute disease, such as measles.

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TABLE OF SPINAL LOCALISATION

Vastus . late- *largely*	*sion at knee.*			*and strong* *t shoulder,* *ward rota-* *; arm is* *s the body;* *t elbow;* *at wrist,* *pronation;* *of fingers;* *wn down;* *tion and* *of neck.*	*None.*	*None.*
Rectus us, Gra- agn. et fasciæ . long. allucis, Glutæus *imental.*	*Adduction at hip; extension at knee; some dorso-flexion at ankle, and some extension of hallux.*	*None.*	*None.*			
terossei, phincter *tal.*						
itor ani, riduals),	*Perineal.*	*Dilator to genitalia and lower rectal muc. membrane.*	*Contraction of bladder; turgor of penis; descending colon and rectum; int. sphinct. ani relaxes.*	*As for D III., but slighter effect, except to lungs, where stronger, vasomotor to hand.*	*Slight opening palp. lass.; secret. Wharton's duct; sweat glands of arm and hand.*	
).	*Perineal.*	..	..	*Slight to vessels of head and face; to forearm and hand (strong), lung; slight acceleration of heart; slight constriction of portal vein.*	*Submaxill. secretion (slight); sweat glands of hand; contract. of spleen.*	

being printed in *italics*, and rest on observations obtained

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AFFERENT ROOT.					No. of Nerve.	
Skin.	Muscle.	Viscera.	Reflex Movement.	Level of Surface Origin.		Striped Muscle.
Lower border of zone runs from eighth dorsal spine to end in front below the attachment of the xiphoid cart. (Head).	Same as motor.	Heart, lungs, stomach (cardiac end), *bile duct and gall bladder.*	..	Ranges from lower edge of second dors. spine to upper of fifth.	D VI.	Same as D V.
Lower border of zone at ninth dors. spine passes to end at junction of upper with middle third of space between xiphoid and umbilicus (Head).	Same as motor.	Heart (auricle), lungs, stomach (card. end), liver, and gall bladder.	*Muscles of back and side of chest; some of the more superficial respond more readily than do those of the intercost. space. Those of the seventh, eighth, and sixth spaces do respond.*	Ranges from top of fourth to bottom of fifth dorsal spine.	D VII.	Erector spinæ, Levator costæ, versospinal, Subcostalis, Interco Obliq. ext. abd. et int. abd., I abd. and Transvers. abd.
Lower border of zone at eleventh dors. spine to end in front at junction of middle with lowest third of space between xiphoid and umbilicus (Head).	Same as motor.	Heart (auricle), lungs, stomach, liver, and gall bladder.	..	Ranges from top of fifth to top of sixth spinous p.	D VIII.	Same as D VII.
Lower border of zone at twelfth dors. spine to end in front at the umbilicus (Head). According to Head the umbilicus lies between ninth and tenth dorsal fields.	Same as motor.	Stomach (pyloric), liver, gall bladder, and intestine.	*Epigastric reflex* (Dinkler).	Ranges from midway between fifth and sixth dorsal spines down to top of seventh.	D IX.	Erect. spin., Lev. cost., Transv Subcostales, Intercost., Oblig. int. abd., Transversus abd., Rect Serrat. post. inf.
Lower border of zone at third lumbar spine to end in front midway down between umbilicus and symphysis (Head).	Same as motor.	Liver, gall bladder, intestine, prostate.	*Dilatation of renal vessels characteristic (Bradford).*	Ranges from lower edge of sixth to upper	D X.	Same as D IX.

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MUSCULAR DYSTROPHIES

Progressive Muscular Atrophy commencing in the Muscle Substance—Pseudo-hypertrophic Paralysis

MUSCULAR DYSTROPHY

In the muscular atrophies so far described the wasting of the muscles is distinctly secondary to disease of the nervous apparatus, from which in the normal state proceed influences necessary for their nutrition. But there remains a group in which the disease appears to start in the muscles themselves, and for which we can find (at least with our present methods of examination) no corresponding lesion in the spinal centres or their connections. In some cases such disease of muscle is characterised mainly by wasting; in some by apparent enlargement; in others by weakness without evident alteration of bulk. And inasmuch as all three conditions appear to be intimately related, and indeed may actually be intermingled in the same patient, the inclusive term "myopathy" or "muscular dystrophy" may be used for the whole group.

Examined anatomically, such muscles are pale and fatty looking; the microscope shows that between the fibres there is generally an interstitial overgrowth either of fat or of fibrous tissue, while the fibres themselves are thinned, and their transverse striation gradually passes over into an appearance of granular degeneration, or perhaps they show waxy changes or vacuolisation. But in some cases of pseudo-hypertrophic paralysis specimens of muscle, removed during life, have shown actual enlargement of the fibres. It is not absolutely certain whether the interstitial overgrowth or the degeneration of the muscle fibres is the primary change; but the first of these views appears to be most commonly held.

The main points which clinically serve to distinguish myopathic atrophies from those of spinal origin are, that in the former—

1. The age at onset is, for the most part, an earlier one.

2. Hereditary influence is often traceable, for it is frequent to find several cases in one family.

3. The point of attack is different; the hands do not suffer first, but rather the large muscles of arm or of shoulder or pelvic girdle. Bulbar paralysis does not occur.

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4. The muscles affected do not correspond, in their grouping, to the grouping of the nuclei in the various spinal segments.

5. Apparent hypertrophy of some muscles may co-exist with atrophy of others; or again there may be congenital absence of some muscles.

6. Fibrillary twitchings are absent as a rule.

7. There is never a typical electrical reaction of degeneration; although the electro-contractility, faradic and galvanic, is likely to diminish and disappear as the muscle wastes.

8. The tendon reactions are not, as in amyotrophic lateral sclerosis, increased; though the knee-jerk may disappear from atrophy of the extensor cruris.

Certain clinical types of myopathic atrophy have been described. One, called the "juvenile type," commences in adolescent life, before the age of twenty; it manifests itself first in the large muscles of the upper arm or shoulder, and the wasting is bilateral, though perhaps not strictly symmetrical. The biceps, triceps, and supinator longus waste; the pectorals, latissimi, and serratus magnus follow. It is noteworthy that the deltoid and spinati do not atrophy along with the biceps and supinator longus, as is usually the case in muscular atrophy of spinal origin. The whole of the trapezius may waste, whereas in spinal atrophy the upper fibres often escape. The paralysis of some muscles produces certain very characteristic effects; thus when the trapezius and serratus are gone, the point of the shoulder drops downwards, and the scapula is suspended by the levator anguli scapulæ from its inner angle. When the serratus is paralysed and the patient raises the arm (by the intact deltoid) in front of him, the lower angle and vertebral border of the scapula stick out like a wing. The forearms commonly escape, and the hands nearly always. The erectores spinæ are not unfrequently affected, and the muscles of the pelvic girdle and of the thigh; indeed the disease may begin here.

Another and less common form is called the "infantile" or the "facio-scapulo-humeral" type. The main difference is that this begins in the face, and in very early life. The orbicularis oculi and orbicularis palpebrarum are affected, so that the eyes cannot be closed completely or forcibly, and the patient cannot whistle nor blow a candle out. The expression of the face is peculiar, but no great inconvenience is caused to the patient, so that the condition may easily pass unrecognised. Later on, however, the shoulder muscles and upper arms become affected. The course of these diseases consists in a very slow progress. As months and

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years go on the affected muscles get worse, and others become involved. Sometimes so many of the muscles are by degrees attacked, that the patient becomes what is popularly known as "a living skeleton." Special dangers of course arise when the disease spreads to the diaphragm or intercostals; trivial pulmonary complaints may then easily assume a grave aspect. Periods of arrest may occur, and may perhaps be induced by such treatment as massage, electricity, and attention to the general health, but no real cure is known.

PSEUDO-HYPERTROPHIC MUSCULAR PARALYSIS

Pseudo-hypertrophic paralysis certainly stands in close relation to myopathic atrophy. Both consist in an idiopathic progressive degeneration of muscle, which manifests itself during life by a progressive paralysis. But while in the disease which we have just considered wasting is the prominent feature, only a few, if any, of the muscles being enlarged, in this disease it is just the reverse, enlargement being a prominent feature. Moreover it begins earlier, in early childhood rather than early adult life, and its progress is more rapid, few of its victims attaining to maturity. Heredity is a common feature in both diseases; but pseudo-hypertrophic paralysis selects as a rule the male members of a family; the females commonly escape themselves, but nevertheless transmit the disease to their offspring. It is said that, when it does attack the female, the disease is less severe and less rapidly progressive than in the male, and that this also is the case in the somewhat rare instances where the disease has begun in adolescent or early adult life.

Symptoms and course. — The first symptom noticed is generally an insecurity in walking; the child easily falls, and cannot go upstairs well. Neither can he run or jump like other children. Walking, standing, and even sitting upright become gradually impossible. Weakness in the upper limbs, chiefly in the shoulders and upper arm muscles, is noticed later. The examination of such a patient will bring out some striking points. Thus in standing or walking he keeps the shoulders and body thrown backwards; this is done to compensate for the weakness of the glutei and erectores spinæ, for while the centre of gravity is thus kept as far backward as possible the patient is less likely to be upset by a slight push forward. Any antero-posterior curvature of the spine thus produced disappears when the patient sits down.

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The gait is peculiar, for in addition to the pose of the body just described there is a peculiar roll from side to side at each step; the feet are generally kept well apart to secure a better basis of support; sometimes there is a "high-stepping" action to clear the toes from the ground; sometimes, on the other hand, owing to weakness of the flexors of the hip, there is difficulty in flexing the thigh and bringing it forward. But the peculiar position of the trunk and the sideward roll are the main features of the patient's walk. If he is placed on his back upon the floor, and told to get up, he does so in a characteristic fashion. He will roll over, get on to his hands and knees, and then, straightening out his knees, and keeping his feet and hands upon the ground, he works his hands backwards, supporting his body by them till he can transfer them to his knees; he next works his hands up his thighs, and so pushes himself up till he can get upright; the object of this manœuvring being to supply the deficiency in the extensors of the hip and erectors of the spine. When stripped the calf muscles are seen to be enlarged, and they are unduly firm to palpation; their bulk often contrasts with that of the thighs above them. The infraspinatus is another muscle that is often enlarged; so too are the deltoids. In some cases this muscular enlargement is so general that the child may look like an "infant Hercules," though his strength is the reverse of Herculean. More often, though the calves and other muscles are enlarged, some are atrophied and some may be congenitally absent: absence of the latissimi dorsi and of the lower part of the pectorals is indeed the rule, so that the axillary folds are ill-marked, and when picked up by the arms, the patient's body seems to slip downwards. The knee-jerks are normal in the earlier stages of the disease, but gradually disappear as the extensor cruris becomes affected. The hypertrophied calves in course of time contract, and thus a talipes equinus is produced, which constitutes a further hindrance to standing and walking. Another deformity frequently seen is a lateral curvature of the spine; this is probably connected with the weakness of the back muscles.

The progress is from bad to worse; gradually walking and standing become impossible; next the back becomes so weak that the patient cannot sit up. Thus before he is grown up the patient becomes bedridden, and dies probably of some intercurrent pulmonary disease. In some cases, however, and particularly in those where the onset has been comparatively late, there is less rapid progress. The best treatment appears to be to enjoin moderate exercise of

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the muscles, while this is yet possible, keeping the patient as long as possible from absolute confinement to chair or bed; passive exercise, with massage and electricity, should be given, and tonic medicines; but the prognosis is in any case bad, and a more rapid end is to be feared than in the other kinds of muscular dystrophy.

It must be remembered that muscular wasting may occur from other causes than those which we have been considering. Mere disuse may cause it, as is well seen in the case of a limb which has been confined in splints. In hemiplegia from cerebral disease (p. 168) there may be some wasting of the hand and upper limb, and this, it would seem, is not due solely to disuse, for such wasting is not always proportionate to the intensity and duration of the hemiplegia. A similar wasting is said sometimes to accompany hysterical paralysis. A well-marked muscular atrophy may accompany disease of a joint. The joint disease is not always extensive, the atrophy picks out certain muscles, and there is often an exaggeration of tendon reactions, so that a suspicion of muscular atrophy from spinal disease may be strongly suggested. The atrophy, however, stands in a definite relation to the affected joint, selecting principally the muscles that extend the joint—the deltoid and spinati in the case of the shoulder, the quadriceps cruris in the case of the knee, the interossei in the case of the finger joints,—and even if it spreads (as is said occasionally to happen) over all the limb, it does not attack the limb of the other side; there is never any electrical reaction of degeneration, nor any electrical change beyond a certain diminution in irritability; lastly, the atrophy can be cured either by successful treatment of the joint itself, or by a course of electricity and massage applied to the muscles.

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MYOTONIA CONGENITA

Syn. Thomsen's Disease

This is a very rare condition, at least in this country It is an affection of the voluntary muscles; their relaxation after a voluntary contraction (and perhaps the initial contraction itself) takes place very slowly, so that attempts at movement are baffled by a tonic spasm. This spasm, if slight, may merely cause a stiffness and awkwardness in action, so that the patient may continue his avocations and even conceal his infirmity from others; but if severe it may be most disabling, as he is liable to be fixed in extremely inconvenient or ridiculous positions. With repeated attempts at the movement the spasm becomes less and less marked, so that the desired action can at length be accomplished. Almost any action, save the semi-voluntary acts of respiration, micturition, defæcation, etc., may be thus impeded, but those of the limbs are most likely to suffer. Thus it may be difficult for the patient to begin walking, to rise suddenly from a chair, to perform the quick movements necessary for dancing or for military drill, or for various handicrafts. Even speaking, or in rare instances swallowing, may be embarrassed. As a rule, only the particular muscles engaged enter into spasm, but a bad spasm tends to make the whole body rigid. The patient's muscles are large and firm, so that he looks athletic; and though they are not strong in proportion to their size, yet there is no progressive enfeeblement, as in pseudo-hypertrophic paralysis. According to Erb, they exhibit an increased irritability to mechanical and electrical stimuli; and the contraction set up by the direct application of strong faradism to a muscle, or still more by the application of a galvanic shock, has a remarkable persistence. The anodal closure contraction is often as readily obtained as the cathodal. Lastly, an uninterrupted galvanic current produces during its passage a series of wave-like muscular contractions which seem to pass from the region of the cathode to that of the anode.

Microscopically the muscular fibres are increased in size, and their nuclei are numerous; their transverse striation is indistinct, and there may even be vacuoles in their substance.

The disease appears to depend upon hereditary influence, for it often runs in families, and generally dates from very early life. Although the sufferers may learn from experience what conditions make them better or worse, no cure is known for the complaint.

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MYASTHENIA GRAVIS

This is a curious paralytic affection, which appears to consist in an exaggeration or over-ready production of muscular fatigue, so that while the affected muscles act normally after rest, after a very slight amount of use they become feeble or incapable of action.

For this disease no morbid anatomy is known, and its etiology is still uncertain; the suggestion has been made that it is due to some toxin, and this finds some justification in the fluctuations of the paralytic phenomena, and in the fact that the complaint has been known to follow various acute diseases. Since the muscles innervated from the pons and medulla are specially liable to the affection, it has also been called "asthenic bulbar paralysis."

The appearance of the patient is often peculiar owing to a droop of the eyelids and an expressionless look produced by weakness of the facial muscles. Sometimes he corrects this ptosis by tilting the head backwards; sometimes the head falls forward from exhaustion of the neck muscles. There may be diplopia, or ophthalmoplegia, or a certain amount of nystagmus. Mastication and swallowing may be difficult. Exhaustion of the respiratory muscles may give rise to alarming dyspnœa, and death may take place from respiratory failure. The trunk muscles may be affected; and the limbs may suffer in various ways, either from a general fatigue, or from fatigue of special muscles. But the essential point is the speedy exhaustion of power, so that muscles which act well at first are soon tired out. Thus the patient may be able to walk, but not for long; he may be able to read aloud, but he soon has to leave off; and he is best in the morning after a thorough rest. The same thing may sometimes be demonstrated electrically; a faradic current, when first applied, causes the usual muscular tetanus; but if the current be maintained, the muscle soon ceases to act. This has been called "the myasthenic reaction." The symptoms are liable to fluctuations; cold, mental emotions, and other factors are said to make them worse. They may disappear entirely, and reappear after a long interval. Not a few cases have terminated fatally.

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LESIONS OF THE CAUDA EQUINA

Anatomy.—The spinal cord in the adult ends at the level of the upper part of the second lumbar vertebra, whilst the spinal dura mater or *theca* extends as a hollow tube as far as the second sacral vertebra. Within the theca is a mass of long spinal nerve roots, anterior and posterior, lumbar, sacral, and coccygeal, concealing in their midst the delicate *filum terminale* of the cord. To this mass the term *cauda equina* is applied. The nerve roots of the cauda equina course so obliquely as to be almost vertical, and perforate the theca, to emerge from their corresponding intervertebral foramina. The filum terminale extends within the vertebral canal beyond the theca as a fine cord which is attached below to the last sacral vertebra or to the upper part of the coccyx.

From the clinical point of view, the cauda equina may be considered simply as a mass of nerve roots, lesions of which will, therefore, produce "root symptoms," motor in the case of the anterior roots, sensory in the case of the posterior, without any symptom of implication of the centres in the spinal cord.

Pathology.—Primary disease of the nerve roots of the cauda is rare. More commonly the lesion is secondary to diseased processes of the spinal meninges or of the bones of the lower lumbar vertebræ or of the sacrum. Traumatic injuries may be produced by fractures of the spinal column below the level of the first lumbar vertebra, by bullet-wounds or stabs, or by hæmorrhages the result of violence insufficient to fracture the bones (as in falls from a height on to the sacral region). Syphilis may attack the nerve roots either in the form of gummata or by causing a chronic meningitis. Various tumours (sarcoma, glioma, angioma, neuroma, etc.) also occur in this region, arising either in the cauda itself or spreading into it from the adjacent bones or meninges. Tuberculous disease of the bones sometimes produces an abscess which compresses the theca. Not uncommonly the nerve roots of the cauda equina are spread out in the sac-wall of a spina bifida.

It will be readily understood that partial lesions of the cauda equina are more common than total ones, and that sometimes the *conus medullaris*, or extreme tip of the spinal cord (including the coccygeal and the two lowest sacral segments) suffers along with the cauda itself.

Symptoms.—These depend upon the precise nerve roots

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involved. In cases where the whole cauda equina is implicated there is flaccid paralysis and atrophy of all the muscles of the lower extremities, anæsthesia of the lower extremities, buttocks, perineum, and genitals, absence of knee-jerks and ankle-jerks, loss of plantar and anal reflexes and paralysis of the bladder and rectum, with loss of sexual power.

If the first lumbar roots escape, as is frequently the case, the anæsthesia is less extensive and spares the inguinal regions and the upper and anterior part of each thigh.

If the lesion is below the third lumbar vertebra, and the third lumbar roots, which give off the anterior crural and obturator nerves, escape, the muscular paralysis and wasting involve only the muscles supplied by the sciatic, gluteal, and pudic nerves, and the result is atrophic paralysis of the glutei and hamstrings, and of all the muscles below the knees, with paralysis of anus and bladder and with anæsthesia in the corresponding root areas. The knee-jerks survive.

If the lesion be still lower down, the muscular paralysis and cutaneous anæsthesia are correspondingly diminished in extent. Below the level of the second sacral roots the resulting area of cutaneous anæsthesia is characteristically "saddle-shaped," including the skin around the anus, the perineum and posterior aspect of scrotum and penis, with a small strip of skin running from the perineum along the postero-internal side of the thighs. The urethral and vesical mucous membranes are also anæsthetic. The bladder and rectum are paralysed, and the anal reflex is lost. There is no muscular paralysis of the lower limbs, and the knee-jerks and plantar reflexes are present.

Lesions of the lowest roots of the cauda equina produce a still more limited paralysis, sparing successively, in passing downwards, the genital apparatus, then the bladder and rectum, which become less and less affected, until finally, with a lesion restricted to the coccygeal nerve, the only symptoms consist in paralysis of the levator ani with anæsthesia of the anus and perineum, the sphincter ani remaining intact.

Diagnosis.—A lesion of the whole cauda equina can usually be readily distinguished from an affection of the spinal cord in the lumbar region, for although the anæsthesia and muscular paralysis in the two cases may be identical in distribution, yet the muscular atrophy, the presence of the electrical reactions of degeneration, and the absence of the knee-jerks, and of the normal plantar, the vesical, and anal reflexes, with constant dribbling of urine, contrast

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markedly with the absence of muscular atrophy, the normal electrical reactions, the increased knee-jerks, the ankle-clonus, the "extensor" plantar reflex, and either the retention or "reflex incontinence" of urine and fæces which are so characteristic of a lesion in the upper lumbar region of the cord. That is, whilst the symptoms are of the same anatomical distribution in the two cases, they are of different types; those of the cauda being of the lower motor neurone, as those of the lumbar cord are of the upper neurone.

It is much more difficult to diagnose a partial lesion of the cauda equina below the level of the third sacral roots from one of the conus medullaris, which is that portion of the spinal cord below the third sacral segment. In both cases the muscles of the lower extremities are unaffected, and there is anæsthesia of the "saddle" area with loss of sexual power and paralysis of the bladder and rectum. Lesions of the cauda, however, tend to be gradual in onset, and are usually accompanied by intense and persistent pains in the sacral region, often with hyperæsthesia of the skin preceding its ultimate anæsthesia. Lesions of the conus medullaris show a rapid onset of motor paralysis and muscular atrophy; there is a greater tendency to the formation of a bed-sore, and the anæsthesia is not preceded by spontaneous pains nor by hyperæsthesia, as in the "root pains" of a lesion of the cauda equina. A partial anæsthesia—analgesia or thermanæsthesia—is also in favour of a lesion of the conus rather than of the cauda. In cases where the conus medullaris is diseased along with the cauda, as not infrequently happens, the symptoms are those of a cauda equina lesion.

Prognosis and treatment.—Compared with affections of the spinal cord, the prognosis in lesions of the cauda equina is somewhat less gloomy, inasmuch as conditions such as syphilitic affections and hæmorrhage cause less permanent damage here and are more frequently recovered from than is the case with similar conditions occurring in the cord itself. Tumours which do not infiltrate but merely compress the nerve roots of the cauda can sometimes be removed surgically, and cases of bone disease in which an abscess presses upon the theca often give excellent results under operative treatment. Syphilitic cases should be treated by iodides and mercury. The question of operation in traumatic cases must be decided upon the particular merits of each individual case, bearing in mind the fact that a hæmorrhage amongst the nerve roots tends spontaneously to become absorbed.

PURVES STEWART.

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ACUTE ASCENDING PARALYSIS

SYN. LANDRY'S PARALYSIS

For purposes of classification, this disease is placed in a special category, intermediate between the affections of the central nervous system and those of the peripheral nerves, partaking of features common to both classes. Thus whilst its clinical course presents many points of resemblance to an affection of the peripheral nerves — some writers, indeed, classify it as a mere variety of peripheral neuritis — yet, on the other hand, the slightness or absence of sensory phenomena, together with the morbid appearances on microscopic examination, indicate that the lower motor neurones are specially picked out as the seat of the disease, the sensory neurones escaping entirely, or almost so. It is probable that several conditions more or less distinct are at present included within this clinical affection.

Etiology.—The disease is much commoner in males than in females, and the majority of cases occur between the ages of twenty and forty years. It is sometimes a sequel of one of the specific fevers; in a few cases syphilis has been an antecedent, whilst in others no previous disease could be ascertained.

Pathology.—For some time this disease was supposed to show no pathological appearances recognisable after death. Now, however, there can be little doubt that it is due to a microbic toxin which expends itself chiefly on the peripheral motor neurones in the spinal cord and nerves, commencing as a rule with those at the lower end of the cord and advancing upwards towards the medulla. This view is supported by the fact that, in addition to evidences of neuritis, distinct changes in the anterior cornual nerve cells have been demonstrated by the Nissl method in cases of the disease.

Symptoms.—The patient, after several days of slight premonitory symptoms, consisting in malaise, headache, pains in the back and tingling of the extremities, develops flaccid paralysis, first in one leg and then, a few hours later, in the other leg. After the legs are completely paralysed, the muscles of the abdomen and of the thorax become successively affected. In a few days the arms are attacked by flaccid palsy, and if the disease ascends higher up the cord, the diaphragm and neck muscles suffer, deglutition

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and articulation become difficult, and even the facial and ocular muscles may become weak. The mental condition of the patient remains perfectly clear to the end. The temperature remains normal throughout; although there is often profuse sweating. Albuminuria may be present. The spleen is frequently enlarged.

In typical cases sensory symptoms are very slight, consisting in slight blunting of sensibility to all varieties of stimuli, chiefly in the extremities. Absolute anæsthesia does not occur. Pains are rare, usually only on pressure of the muscles. There is neither atrophy nor any marked electrical change in the paralysed muscles. The knee-jerks and superficial reflexes are lost. The bladder and rectum generally remain unaffected.

The majority of cases are fatal. Death may occur within two or three days from asphyxia due to paralysis of the respiratory muscles, or may not supervene for a week or even longer.

In less severe cases the disease may become arrested, and, after a stationary stage, recovery slowly sets in, beginning in those muscles which were the latest to be attacked, the legs recovering last of all.

Treatment must be on general principles. Diaphoresis should be encouraged by warm baths or vapour baths. Serum-therapy has not yet yielded the desired antitoxin which might be administered. Several cases, however, are recorded in which recovery followed the administration of ergotin. Counter-irritation to the back, as by a long narrow mustard plaster or by the actual cautery, has been recommended. The patient should, of course, be kept at absolute rest on a water-bed.

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DISEASES OF THE PERIPHERAL NERVOUS SYSTEM

The peripheral, as distinguished from the central nervous system, includes within its territory all neurones, efferent or afferent, which extend beyond the central cerebro-spinal axis. The efferent neurones are those whose trophic centres are in the anterior cornua of the spinal cord, or in the homologous gray matter of the medulla or pons; whilst the afferent neurones are those which have their trophic centres in the ganglia of the posterior spinal roots, or, in the case of the trigeminal nerve, in the Gasserian ganglion. Under this category, therefore, are included the cranial and spinal nerves. The sympathetic nervous system with its association neurones will also be described under this heading.

Those spinal nerves which have a considerable distance to traverse from the spinal cord before they emerge from the spinal canal, viz. the nerve roots of the cauda equina, have already been described (p. 218), since from the clinical standpoint lesions of the cord are the chief conditions from which they have to be diagnosed.

Affections of the peripheral nervous system differ from those of the brain and spinal cord both in kind and in degree. The most striking difference in kind is exemplified in the fact that "functional" diseases, in the accepted sense of the term, do not primarily affect the peripheral nerves but are confined to the higher cerebral centres, the peripheral nerves being only secondarily implicated in so far as they act as channels through which central symptoms are manifested. The pain of some varieties of neuralgia, which on superficial observation might seem to fall within the category of "functional" affections of the peripheral nerves, does not really belong to that class, for pain implies the existence of something more than a peripheral conducting system, it postulates a centre, a sensorium. Indeed we may go farther and maintain that the pain in many of the most severe cases of neuralgia appears to be really of central origin, since it sometimes persists even after the painful area of skin has been rendered anæsthetic by division of its afferent nerves. In other cases of neuralgia there exists some local physical cause which excites the pain. But such

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cases can hardly be considered as within the strict category of "functional" diseases.

There are several important anatomical points of difference between the peripheral nerves and the central nervous organ. Not only are the axis cylinders, as a rule, greater in length in the peripheral nerves, but they have an additional connective-tissue element superadded, which is intimately associated with them in the form of a nucleated neurilemma around each axis cylinder and a perineurium of fibrous tissue binding together bundles of nerve fibres into firm cords. The length of the peripheral nerves, especially of the limbs, and the presence of an abundant connective-tissue element are two conditions of the greatest importance as determining factors in disease. The length of the peripheral nerves not only exposes them to traumatism in exposed situations, but also renders them liable to become implicated in many diseases of adjacent tissues and organs, even more markedly than in the case of the brain and spinal cord, which are to such a large extent shut off from other organs and by their deep situation are less exposed to traumatism. Affections of the peripheral nerves due to traumatism or secondary to disease of adjacent organs or tissues are habitually irregular in distribution and only in exceptional cases do they approach to any appearance of symmetry. In this respect they are unlike traumatic cerebral or spinal lesions, which tend to produce symptoms either hemiplegic or paraplegic in distribution.

Amongst other causes, apart from traumatism, which affect the peripheral nerves in virtue of their length, may be mentioned weakening of central trophic influence. General marasmus and malnutrition, senile and cachectic conditions and exposure to cold may be adduced as examples of causes which for this reason affect the peripheral more than the central nervous system. The perverted metabolism of such a disease as diabetes probably affects the peripheral nerves in a similar fashion, those parts at the periphery being the first to show evidence of weakening of the central trophic influence.

In connection with the anatomical length of the peripheral nerves may be mentioned the effects of exposure to various toxic agents, whether microbic or non-microbic in origin. As a rule, such poisons, circulating as they do in the blood-stream, affect the peripheral nerves symmetrically on both sides of the body. And although it is true that the central nervous system is as much exposed to the circulating toxic agent as are the peripheral nerves,

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yet the distal ends of these long nerve trunks which are presumably less resistant, from being farthest from their trophic centres in the anterior cornua and posterior root ganglia respectively, suffer earlier than the centres themselves. Some poisons have a selective affinity for certain neurones; thus the diphtheritic toxin attacks especially the peripheral nerves of the soft palate and ocular muscles, whilst lead has a special preference for certain of the nerves of the upper extremities. The special affinity for the cells of the anterior cornua exhibited by strychnine may be recalled in this connection.

The presence of a fibrous element, in the shape of the neurilemma and of the perineurium, is also of considerable pathological importance, since certain diseases which attack fibrous structures may therefore affect the peripheral nerves to a much greater extent than the central nervous system. As examples of this may be mentioned such diseases as gout and rheumatism, which cause interstitial peripheral neuritis with comparative frequency and yet produce neither encephalitis nor myelitis.

The diagnostic characters of peripheral as contrasted with central lesions hardly require to be alluded to at this stage. Suffice it to remind the reader of the muscular flaccidity and atrophy, of the changes in the electrical reactions in the affected muscles, and of the diminution or loss of the deep reflexes which are so characteristic of lesions of the lower motor neurones, and of the anæsthesia or other varieties of sensory impairment which, in addition to certain peculiar trophic changes in the skin, nails, etc., occur in affections of the lowest sensory neurones.

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AFFECTIONS OF THE CRANIAL NERVES

I. OLFACTORY NERVES

Origin and distribution.—The olfactory nerves—about twenty on each side—arise from the under surface of the olfactory bulbs and pass through the cribriform plate of the ethmoid bone to be distributed to the upper part of the nasal mucous membrane, where they are connected with bipolar cells, from which terminal filaments spread towards the surface. The olfactory tract passes backwards from the olfactory bulb, and its fibres may be traced to the uncinate gyrus of the same side, towards the optic thalamus and internal capsule, and to the opposite side *vid* the anterior commissure, which in part connects the hippocampal lobules of both sides.

The olfactory nerves may be congenitally absent. They may be destroyed or damaged by various diseased conditions in the floor of the anterior cranial fossa, or they may be ruptured by fractures of the skull implicating the cribriform plate. In some cases of tabes the olfactory nerves undergo atrophy. Severe intracranial pressure, from whatever cause, may compress the olfactory bulbs. *Hyposmia*, or diminution of smell, and *anosmia*, its total loss, are the result.

Much more commonly, however, anosmia is due to changes in the nasal mucous membrane, whether from catarrh, nasal polypi, trophic changes, or new growths.

Parosmia, or perverted sense of smell, has been noticed in morbid states involving the cortical olfactory centres.

II. OPTIC NERVE

Origin and distribution.—The optic nerves run forwards from the optic commissure, where partial decussation of the optic tracts occurs. Thence each nerve passes through the optic foramen of its own side, to enter the eyeball, where it spreads out as the retina. The fibres of the optic tract are to be traced backwards from the chiasma, to end in the pulvinar of the optic thalamus, the external geniculate body, and the superior quadrigeminal body. These ganglia are connected by the "optic radiations" with the occipito-angular region of the cortex.

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The optic nerves may become pale and atrophic, either primarily, as in tabes and in insular sclerosis, or secondarily, after a preceding stage of optic neuritis. Optic neuritis may be due to numerous conditions, chief amongst which are intracranial tumours, renal disease, and lead poisoning. But it may also occur in chlorosis, leukæmia, pernicious anæmia, and diabetes mellitus (*see* p. 390).

It must not be forgotten that optic neuritis may attain a degree of considerable severity without the acuity of the patient's vision becoming impaired. But as the neuritis progresses towards atrophy, vision gradually grows less acute, the visual field in the affected eye becomes concentrically contracted, whilst the colour fields shrink concentrically and ultimately disappear, the field for green being the earliest affected, therein contrasting markedly with hysterical amblyopia. Ultimately, as the atrophy becomes complete, all perception of light is lost, in one or both eyes according as the atrophy is unilateral or bilateral.

LESIONS OF THE OPTIC CHIASMA may here be conveniently referred to. Tumours and syphilitic or inflammatory affections of the bones of the anterior cerebral fossa, of the brain or meninges, or of the pituitary body, may cause damage to the chiasma.

If the lesion be in the central part of the chiasma, interfering with the decussating optic fibres, which belong, it should be remembered, to the nasal halves of both retinæ, vision is lost in the outer portion of each visual field—*bi-temporal hemianopsia.* If, again, the lesion be situated at one or other lateral extremity of the chiasma, it will interfere only with the direct, non-decussating fibres of the optic nerve and tract of the same side, which correspond to the temporal half of that retina, the result being *unilateral nasal hemianopsia* in the eye of the corresponding side. Two such separate lesions, one on each side, will of course cause a *bilateral nasal hemianopsia.* A lesion involving the central part of the chiasma, and extending into one of its extremities, produces the sum of those two, namely bi-temporal hemianopsia *plus* unilateral nasal hemianopsia, that is to say, total blindness of one eye with temporal hemianopsia of the other. Still more extensive disease, involving the whole of the chiasma, interrupts the fibres of both optic nerves, and total blindness is the result (*vide* Fig. 6, p. 50).

III. IV. AND VI. MOTORES OCULI

Origin and distribution.—The third nerve takes origin in groups of cells forming the gray matter of the upper (anterior) part of the floor

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of the aqueduct of Sylvius. The fibres thence pass through the tegmentum of the crus cerebri to reach the surface on the inner side of the latter, immediately above the pons.

The nucleus of the fourth nerve is almost continuous with the lower end of the foregoing, being separated by a small group of cells which has been supposed to be the special nucleus for the fibres for the levator palpebræ muscle. From this origin the fibres pass downwards and backwards, round the lower part of the aqueduct, into the superior medullary velum, in which the nerves of opposite sides completely decussate, ultimately to emerge at the surface at the outer side of the cerebral peduncles, in the angles between them and the upper border of the pons.

The nucleus of origin of the sixth nerve lies in the floor of the fourth ventricle, at some little distance below those of the fourth and third, these three being connected by the posterior longitudinal bundle, an arrangement which permits the conjugate movements of the eyes. Passing almost directly forwards, the fibres reach the surface at the lower margin of the pons, directly external to the anterior pyramid of the medulla.

From these several superficial origins the nerves are directed forward, lying in the outer wall of the cavernous sinus, through the sphenoidal fissure, to the orbit, the sixth or abducens being distributed to the external rectus only, the fourth or trochlear to the superior oblique only, and the third to the other extrinsic muscles of the eyeball, to the levator palpebræ superioris, and through the ciliary ganglion supplying the sphincter iridis and ciliary muscle.

The cortical connection of these nuclei is probably effected by fibres which pass downwards through the crus cerebri and anterior limb of the internal capsule from the posterior end of the second frontal gyrus.

PARALYSIS OF OCULAR NERVES—**Causes.**—The chief intracranial causes of paralysis of the third, fourth, and sixth cranial nerves between their nuclei and their entrance into the sphenoidal fissure are new growths and chronic inflammatory conditions, syphilitic or tuberculous, in the bones, meninges or brain substance in the immediate neighbourhood. Of these the commonest cause is syphilis. Increase of general intracranial pressure, apart from focal lesions, may also cause ocular paralysis. The long intracranial course of the sixth nerve renders it more prone to pressure than the third or fourth nerves.

Between the sphenoidal fissure and their motor distribution in the ocular muscles the nerves may be paralysed by orbital growths, by hæmorrhage, whether spontaneous or due to head injuries, and by exposure to cold—so-called rheumatic neuritis. Syphilis, tabes

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and disseminated sclerosis may also cause paralysis, partial or complete, of any of the ocular nerves, whilst post-diphtheritic paralysis is not uncommon in the third and sixth nerves.

Transient ocular palsy, especially affecting the third nerve, sometimes occurs without apparent cause at regular intervals of weeks or months, passing off completely between the attacks. Such attacks are generally associated with headache, most intense in the eye and forehead of the affected side, and with vomiting (Charcot's *migraine ophthalmoplegique*). Sometimes blunting of sensation has been observed in the area of distribution of the first division of the fifth nerve. The etiology of such cases is obscure.

A congenital weakness of certain ocular muscles sometimes exists, affecting most frequently the levator palpebræ superioris or the superior rectus. In such cases of *congenital ptosis* the remarkable fact is observed that although the patient cannot voluntarily raise the upper lid, yet it is elevated whenever certain movements of the jaw are made, particularly when the external pterygoid muscle of the same side is thrown into action.

Symptoms.—Paralysis of any of the ocular nerves causes giddiness, diplopia, and squint, with deficient movement of the affected eye towards the direction of traction of the particular muscle paralysed.

Paralysis of the third nerve—**Symptoms.**—Complete paralysis of this nerve affects all the external ocular muscles except the superior oblique and the external rectus. It also affects the levator palpebræ superioris, the sphincter pupillæ and the ciliary muscle. As a result there is ptosis with over-action of the corresponding half of the frontalis muscle, so that the eyebrow stands higher than it ought to be. The eye cannot be rotated upwards, directly inwards, or directly downwards, although a slight downward and inward movement can be executed by the superior oblique. The pupil is of medium size, and does not contract to light. The power of accommodation is lost. Later, the external strabismus becomes more marked, from contracture of the unopposed external rectus, and the pupil, from a similar contracture of the dilator pupillæ, becomes more dilated. Total paralysis of the third nerve is relatively rare; partial paralysis, affecting one or more branches, is much more common.

The symptoms in partial paralysis vary according to the muscle or muscles affected. A simple rule applying to all cases of paralytic squint is that the affected eye is displaced in the direction opposite to the direction of traction of the paralysed muscle, whilst the false

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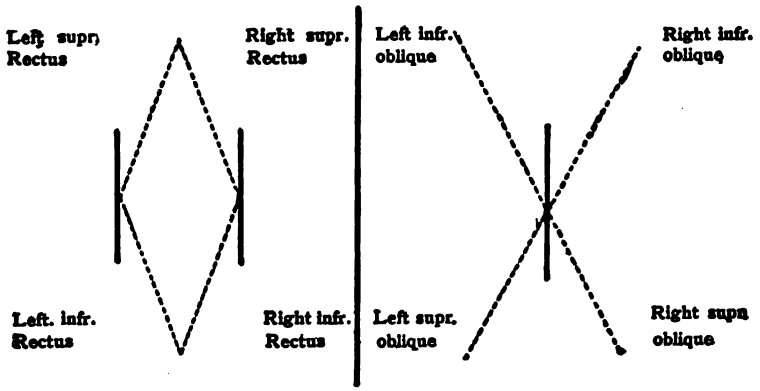
image, seen by the affected eye, is displaced in the direction of traction of the paralysed muscle.



FIGS. 23 and 24.—Dr. L. Werner's "artificial memory" for the double images in ocular paralyses (*Swanzy's Handbook*, p. 513). Fig. 23 shows the position of the images in paralysis of the recti muscles; Fig. 24 in paralysis of the oblique muscles. The dotted lines indicate "false," the continuous lines "true" images. One instance will illustrate the method of using the "artificial memory." Suppose there is paralysis of the *right superior oblique* muscle. Fig. 24 shows that (1) the false image is on the same side as the true; (2) the false image is at an inclination to the true; (3) the false image extends lower than the true; (4) the diplopia occurs on looking downwards.

PARALYSIS OF THE FOURTH NERVE—**Symptoms.**—The fourth nerve supplies only one muscle, the superior oblique, which rotates the eye downwards and inwards. The defective movement is sometimes difficult to detect and the paralysis is recognised chiefly from its characteristic diplopia. In the whole of the field above the horizontal plane there is single vision, but diplopia appears on looking downwards and is most marked when looking downwards and inwards. The false image stands at a lower level than the true, with its upper end inclined inwards towards the other. The false image also seems to the patient to be nearer than the true, the explanation of this being obscure. Clinically the patient feels giddy and sees double when looking downwards, as for example in walking downstairs, and he inclines his head habitually forwards and to the sound side.

PARALYSIS OF THE SIXTH NERVE—**Symptoms.**—The sixth nerve supplies the external rectus muscle alone. Its paralysis is usually easy of diagnosis. There is inability to move the eye outwards, with diplopia on looking in that direction. In old cases with contracture of the unantagonised internal rectus, there is internal strabismus of the affected eye.

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A LESION OF THE SIXTH NUCLEUS causes, in addition to paralysis of the external rectus of the same side, weakness of the internal rectus of the opposite eye during attempted conjugate movement of both eyes towards the affected side. That the affection of the opposite internal rectus is not due to a concomitant affection of the third nerve is shown by the fact that convergence by both internal recti is unimpaired. From the close proximity of the root of the facial nerve, which loops round the sixth nucleus within the pons, a lesion of the latter is often accompanied by facial paralysis on the same side.

Nuclear Ophthalmoplegia. — This is a term applied to ocular palsies of nuclear origin; and although the subject is not, strictly speaking, included in the category of affections of the peripheral nervous system, yet it is conveniently considered along with ocular palsies of infra-nuclear origin.

In cases of nuclear ophthalmoplegia where the internal ocular muscles (*i.e.* the ciliary muscle, the sphincter pupillæ and the dilator pupillæ) are alone paralysed, the condition is one of *ophthalmoplegia interna*, and the signs are those of loss of the power of accommodation with mydriasis and immobility of the pupil. Such paralysis may exist in one or both eyes, and, if bilateral, may be equal or unequal in degree on the two sides. *Ophthalmoplegia externa* is a nuclear palsy affecting, with more or less completeness, the external muscles of the eyeball, usually on both sides, but not necessarily equally in both eyes. It is often associated with ophthalmoplegia interna, but may occur alone. The conjunction of the "internal" with the "external" variety constitutes *total ophthalmoplegia*, in which the eyeballs are fixed and motionless, and the movements of the pupils are lost.

The diagnosis of nuclear ophthalmoplegia from ocular paralysis due to affection of the ocular nerves below their nuclei is sometimes difficult. In doubtful cases, if, in addition to the other ocular muscles, the orbicularis palpebrarum is found to be affected, the lesion is a nuclear and not an infra-nuclear one.

Causes. — Nuclear ophthalmoplegia is generally a chronic disease; sometimes it is subacute or acute. Chronic progressive ophthalmoplegia is analogous to chronic anterior poliomyelitis of the cord (*q.v.* p. 208). It may occur as the last stage of an upward spreading bulbar paralysis; occasionally, however, though less frequently, it commences above in the oculo-motor nuclei and spreads downwards in the gray matter of the bulb. The acute form of ophthalmoplegia (polio-encephalitis superior) is due to a

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hæmorrhagic or thrombotic softening of the oculo-motor nuclei. It is commonest in alcoholic patients, but may be excited by various other poisons, either microbic (as diphtheria, influenza, syphilis), or metallic, such as lead. These acute cases are not infrequently associated with optic neuritis (*see* p. 130).

(For a discussion of the various affections of the pupil *see* p. 386).

Other Varieties of Ophthalmoplegia.—Ophthalmoplegia, or impairment of ocular movements, may, of course, arise from a lesion in any part of the motor tract from the cortex to the oculo-motor nuclei and from the latter to the ocular muscles. The effects of destructive or of irritative lesions on the second frontal gyrus are referred to elsewhere (*vide* p. 117), the former producing paralysis, the latter producing spasm of the muscles which cause conjugate deviation of the eyes to the opposite side. The temporary paralysis of conjugate movement in cases of cerebral apoplexy is also discussed under the latter disease (*vide* p. 69).

V. THE TRIGEMINAL NERVE

Origin and distribution.—The fifth or trigeminal nerve is composed of two distinct parts: a sensory division, arising in the Gasserian ganglion and terminating in the medulla oblongata and pons; and a motor division, springing from two nuclei in the pons and crura cerebri. After emergence from the pons, the motor root lies beneath, and towards the mesial side of the sensory. Both roots course over the apex of the petrous part of the temporal bone, through a lymph space—the cavum Meckelii—between the layers of the dura mater. In this situation lies the Gasserian ganglion. From its anterior end three branches of the nerve are traced. The first division passes in the outer wall of the cavernous sinus to the sphenoidal fissure; the second and third divisions pass through the foramen rotundum and foramen ovale respectively. The motor root courses beneath the Gasserian ganglion, and joins the third division of the nerve as it passes through the foramen ovale. The first and second divisions of the nerve, therefore, are entirely sensory; the third division is a mixed nerve.

The two roots have separate origins. For the motor root there are two nuclei, one in the upper part of the floor of the fourth ventricle internal to the upper end of the "sensory nucleus," the other being a group of large nerve cells at the side of the aqueduct of Sylvius, the fibres from which pass downwards (the descending or mesencephalic root of the fifth nerve) to join those from the other nucleus. The sensory fibres terminate in a lengthy tract of cells extending from a point ex-

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ternal to the lower motor nucleus down through the pons and medulla, forming the substantia gelatinosa of Rolando. The cerebral relations of the sensory end-nucleus are doubtful. To the motor nuclei fibres pass *viâ* the genu of the internal capsule from the cortex.

The first or ophthalmic division passes through the sphenoidal fissure into the orbit. At its exit from the Gasserian ganglion it receives fibres from the sympathetic, which pass with it to the eye, through the lenticular ganglion, to innervate the dilator pupillæ muscle. The ophthalmic division of the fifth nerve is the sensory nerve of the skin of the forehead, upper eyelid, and front of the scalp as far as the vertex, the mesial part of the skin of the nose as far as the tip, the eyeball, the lachrymal gland, the conjunctiva of the upper lid and the mucous membrane of the anterior and upper part of the nasal cavity.

The second or superior maxillary division passes from the Gasserian ganglion through the foramen rotundum across the spheno-maxillary fossa to the infra-orbital canal. In the spheno-maxillary fossa it is connected with Meckel's ganglion, which gives off, amongst other branches, the Vidian nerve. This latter runs backwards to join the facial nerve, the posterior end of the Vidian nerve being called the great superficial petrosal. The superior maxillary division supplies the skin of the cheek, the upper lip, the side of the nose, the lower eyelid, and the anterior part of the temple, the conjunctiva of the lower lid, the upper teeth, the mucous membrane of the upper lip, upper part of the cheek, upper jaw, hard and soft palate, uvula, tonsil, upper part of pharynx, part of the nasal mucous membrane, and that of the middle ear. It also contains some taste fibres.

The third or inferior maxillary division is a mixed nerve which emerges from the foramen ovale. It contains all the fibres of the motor root. These are distributed to the masseter, temporal, and both pterygoid muscles, also to the tensor tympani, the mylo-hyoid and the anterior belly of the digastric. The sensory fibres supply the skin of the posterior part of the temple, part of the outer ear, the lower lip, chin, and lower part of the cheek, the lower teeth and gums, the tongue, the floor of the mouth, and the salivary glands. This division of the nerve probably contains taste fibres at its origin from the Gasserian ganglion.

The course of the taste fibres is complicated. Those for the anterior two-thirds of the tongue reach it through the lingual nerve, which is a branch of the inferior maxillary division. But there is strong evidence to prove that these fibres actually enter the brain through the second division of the fifth, their course being from the lingual nerve to the chorda tympani, running in the aqueduct of Fallopius along with the facial nerve as far as the geniculate ganglion, thence by the great superficial petrosal (Vidian) nerve to Meckel's ganglion, from which the fibres enter the second division of the fifth.

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The taste fibres for the posterior third of the tongue and the palate are distributed in the glosso-pharyngeal nerve, but there is no evidence to show that the glosso-pharyngeal contains any taste fibres at its point of exit from the medulla. On the contrary, it is highly probable that all the taste fibres enter the brain through the Gasserian ganglion, for when the fifth nerve is divided by operation in man, above the Gasserian ganglion, although the glosso-pharyngeal nerve remains

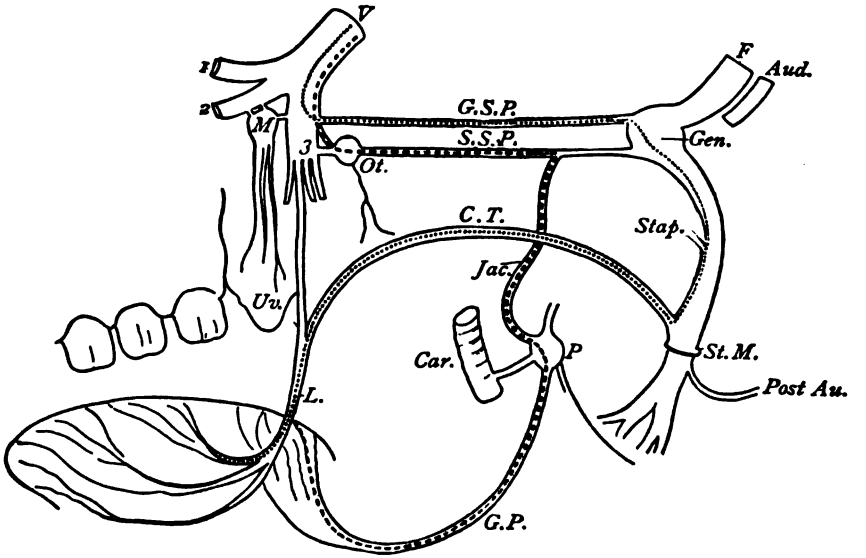

FIG. 25.—Diagram of Facial Nerve and its connections (modified from Leube), showing course of taste fibres. *V.*, trigeminal nerve; 1, 2, 3, first, second, and third divisions of trigeminus; *F.*, facial nerve; *Aud.*, auditory nerve; *G.S.P.*, great superficial petrosal; *S.S.P.*, small superficial petrosal; *Gen.*, geniculate ganglion; *Ot.*, otic ganglion; *M.*, Meckel's ganglion; *Uv.*, uvula; *L.*, lingual nerve; *C.T.*, chorda tympani; *Stap.*, nerve to stapedius; *St. M.*, stylo-mastoid foramen; *Post. Au.*, posterior auricular nerve; *G.P.*, glosso-pharyngeal nerve; *P.*, petrous ganglion of glosso-pharyngeal; *Jac.*, Jacobson's nerve; *Car.*, tympanic plexus near carotid artery.

N.B.—Dotted line = taste fibres from anterior two-thirds of tongue; dash line = taste fibres from posterior third of tongue.

untouched, there is total loss of taste on the whole of the affected side. The route taken by the fibres for the back of the tongue and the palate is probably from the glosso-pharyngeal nerve through its tympanic branch (Jacobson's nerve) to the small superficial petrosal, and thence through the otic ganglion to the third division of the fifth nerve (*see* Fig. 25).

The sense of taste is best examined by rubbing upon the protruded tongue various substances, preferably in fine white powders. The

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patient must keep his tongue protruded until he feels the taste, when he should make a sign. If he be allowed to withdraw the tongue whilst waiting for the sensation to arrive, fallacy may occur from the rolling of the tongue within the mouth and from the flow of saliva to and fro. For the sense of sweetness sugar is the best test, for bitterness, quinine; for the saline taste, common salt, and for the acid taste, citric acid. Volatile oils, such as those of mustard, cloves, peppermint, etc., should not be used, as they stimulate the olfactory and even the nerves of common sensation. A feeble galvanic current, which produces a coppery or metallic taste, is an excellent means of mapping out areas of taste.

Paralysis of the fifth nerve—**Causes.**—Primary isolated disease of the fifth nerve is rare, if we except trigeminal neuralgia. So-called "rheumatic" neuritis of the nerve has been observed, also its paralysis in the course of disseminated sclerosis, tabes, and syringomyelia. At the base of the skull the nerve may be affected by new growths, by gummatous neuritis and by chronic meningitic processes. A comparatively small lesion in the region of the apex of the petrous part of the temporal bone will implicate both sensory and motor roots. Below the Gasserian ganglion the nerve becomes so widely distributed that its branches may be implicated by disease in very varied structures. Caries of the sphenoid bone may cause widespread paralysis of the fifth nerve, and fractures of the base of the skull may affect one or more divisions of the nerve. The ophthalmic division, as it courses along the wall of the cavernous sinus, may be implicated by tumours in the region of the pituitary body, or by aneurism of the internal carotid, whilst in the orbit it may be injured by cellulitis or by new growths. Tumours of the parotid gland and of the neighbouring bones not infrequently attack the second and third divisions of the nerve, at or below the spheno-maxillary fossa.

Symptoms.—Complete paralysis of the fifth nerve produces sensory, motor, and secretory changes in the trigeminal area. The anæsthesia affects the corresponding half of the anterior half of the scalp and the face, there being an area of skin near the angle of the jaw where the anæsthesia is only partial (the cervical nerves partially supplying this area). There is complete anæsthesia of the cornea and conjunctiva and of the mucous membrane of the nose, mouth and tongue. Since this defect extends exactly to the middle line, the patient when drinking feels as if his cup were broken. Taste is completely lost on the affected side, both in front and at the back.

All the muscles supplied by the motor division become

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paralysed and atrophied, and show the electrical reactions of degeneration. There is marked hollowing of the temporal fossa; the zygoma is unnaturally prominent, and when the patient clenches his teeth the masseter muscle cannot be felt to contract on the paralysed side. On opening the mouth the lower jaw swings over towards the paralysed side. This is owing to paralysis of the external pterygoid, which fails to draw the condyle of the jaw forwards on the affected side. Paralysis of the tensor tympani is said to cause difficulty in hearing notes of low pitch, but this is not easy to determine.

The secretion of tears on the paralysed side is diminished, also the secretion of nasal mucus and of saliva. As a result the mucous membranes become abnormally dry, and may show secondary trophic changes. Stimulation of the mucous membrane no longer induces sneezing. Smell at first is unimpaired on the paralysed side, but from dryness and trophic changes in the Schneiderian membrane, there is ultimately complete anosmia in the affected nostril. The corneal reflex is absent, and the tongue is much more furred on the paralysed side than on the healthy side, probably from deficient friction by food on the anæsthetic side. The teeth on the paralysed side are anæsthetic and tend to drop out. This has been ascribed to a trophic change, but is more probably to be regarded as really traumatic, the patient biting clumsily with his anæsthetic teeth. The palatal reflex is abolished on the anæsthetic side.

True trophic changes in the sensory area of the fifth nerve in trigeminal paralysis are the exception rather than the rule. The so-called "neuroparalytic" ophthalmia, which was formerly supposed invariably to accompany complete paralysis of the ophthalmic division, does not necessarily occur. When it does ensue, it is probably due to some irritant gaining access to the anæsthetic eye. Herpes zoster may affect the cutaneous area of one or more divisions of the fifth nerve, but it usually indicates some irritative condition in the Gasserian ganglion or its neighbourhood. Trophic changes, therefore, when present, indicate a neuritic rather than a neuro-paralytic affection of the nerve.

Progressive facial hemiatrophy may here be referred to. It is a rare disease, which comes on usually in childhood or youth and advances very slowly. It is commoner in the female sex than in the male. It may arise without apparent cause, or it may follow some blow or injury to the face.

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Symptoms.—It shows itself first in the skin of the face, either near the orbit or over the upper or lower jaw, and gradually spreads over the whole of that side of the face. The skin becomes thinned; it may be either abnormally pale or pigmented and its subcutaneous fat disappears. The affected side of the face becomes deeply wrinkled and furrowed, contrasting markedly with the normal side. The disease is sharply limited by the middle line. The bones, cartilages and muscles become smaller on the affected side; the teeth may be small, and occasionally the corresponding half of the tongue and sometimes of the soft palate is distinctly wasted. There is no sensory paralysis, nor is there any real motor palsy, and the electrical reactions of the muscles remain unchanged. The pupils may be unequal, that on the affected side being larger or smaller than its fellow. The hair of the beard on the affected side sometimes falls out or becomes white, and the sebaceous glands atrophy. The affection rarely extends up to the scalp. The disease progresses for many years, but does not exercise any injurious effect on the general health. Some cases are preceded by local sclerodermia or associated with sclerodermia in other nerve areas of the body.

The real nature of the disease is still disputed. Some authorities (Möbius, Oppenheim) favour the view that it is a vasomotor affection of sympathetic origin. Virchow, Mendel, Hutchinson, and others ascribe it to an affection of the fifth nerve, possibly in the nucleus of the so-called "descending" root. In one case interstitial neuritis was found in the branches of the trigeminal nerve (see p. 371).

VII. THE FACIAL NERVE

Origin and distribution.—The seventh or facial nerve arises from a nucleus in the lower part of the pons, but it also receives fibres from the nucleus of the third nerve (*vid* the posterior longitudinal fasciculus) for the orbicularis palpebrarum and possibly from the hypoglossal nucleus for the orbicularis oris. Within the substance of the pons the fibres of the facial form a loop which curves round the nucleus of the sixth nerve and the facial nerve finally emerges between the olive and restiform body in close proximity to the auditory nerve. It accompanies the auditory nerve to the internal auditory meatus, there enters the tortuous aqueduct of Fallopius, and passes out from the skull at the stylomastoid foramen. At the geniculate ganglion, which is situated on the nerve at the bend near the upper end of the Fallopian aqueduct, the facial is joined by the great superficial petrosal from the Vidian nerve (containing taste fibres) and by the small superficial petrosal from the otic ganglion. Within the aqueduct the nerve gives off a

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branch to the stapedius muscle, and lower down the chorda tympani leaves it to join the lingual nerve. On emerging from the stylomastoid foramen, the facial gives off its posterior auricular branch, supplying the occipital part of the occipito-frontalis and the muscles of the pinna. The main trunk then divides into numerous branches supplying all the muscles of the face (except the levator palpebræ superioris). It also supplies the frontalis part of the occipito-frontalis, the platysma, the stylohyoid and the posterior belly of the digastric.

The cortical centres for the face are situated at the lower end of the ascending frontal and ascending parietal convolutions. The fibres from this region pass through the internal capsule immediately behind the genu and thence on the inner side of the crus to the nucleus in the pons.

Facial Paralysis.—Of all the palsies of isolated peripheral nerves, that of the facial is the commonest. The facial nerve may be attacked by disease in the substance of the pons, at the base of the brain, in the bony aqueduct of Fallopius, or after its exit from the stylomastoid foramen.

Causes.—Nearly three out of every four cases of facial palsy follow exposure to cold, producing a "rheumatic" neuritis which attacks the nerve just within the stylomastoid foramen. In the Fallopian aqueduct the nerve may also be affected by middle-ear disease, by disease of the temporal bone, by fracture of the base of the skull, and by spontaneous hæmorrhages, as in purpura and other "bleeding diseases." Facial paralysis may also occur in gout, syphilis, tabes and disseminated sclerosis. At the base of the skull meningitis, tumours and aneurisms may attack the nerve, and when in such a situation they usually also affect the adjacent auditory nerve. Malignant tumours in the parotid region may press on the branches, whilst the nerve may also be compressed by the midwifery forceps during birth or divided by wounds in later life.

Bilateral facial palsy is rare. It may be due to intra-cranial or extra-cranial causes. The commonest intra-cranial cause is syphilitic disease at the base of the brain, but fractures of the base or lesions in the pons may also produce bilateral paralysis of the facial. Double otitis media, exposure to cold, and post-diphtheritic paralysis are the most important extra-cranial causes. Alcoholic neuritis rarely involves the face, but, when it does so, the affection is always bilateral.

The **symptoms** of facial paralysis vary somewhat, according to the seat of lesion :—

(A) The nerve may be affected *after its exit from the stylomastoid*

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foramen (Bell's paralysis). In such cases there is facial asymmetry even when at rest, and still more marked on movement. This asymmetry is more evident in old than in young patients. On voluntary or emotional movement of the face, the healthy side moves alone. On the paralysed side the furrows of the forehead are smoothed out and the patient cannot frown or wrinkle the brow on that side. The eye remains widely open, and the tears run down over the cheek instead of into the lachrymal duct. When the patient attempts to close the affected eye, he merely rolls the eyeball upwards until the cornea gets under shelter of the upper lid. This inability to close the palpebral aperture renders the eye liable to irritation and inflammation by dust or other foreign bodies which gain access to it. The conjunctival reflex, of course, is abolished. The tip of the nose is drawn over somewhat towards the sound side, and the naso-labial fold on the affected side is flattened out. There is no true respiratory movement of the ala nasi, even on deep inspiration or on sniffing, although the breath current may mechanically cause the nostril to flap loosely. The mouth is pulled over towards the sound side, and its angle droops on the affected side, from which saliva may dribble. The patient is unable to whistle, and the articulation of labial consonants is impaired. On smiling or showing the upper teeth, the healthy side moves alone. During mastication food accumulates between the jaw and the cheek on the paralysed side, and during expiration the cheek flaps loosely. In the few individuals who have the power of voluntary movement of the pinna, this power is lost. Voluntary movement of the integument of the neck by the platysma is also abolished.

(B) If the nerve be affected *within the Fallopian aqueduct*, it produces all the symptoms described under (A), with, in addition, loss of taste (*ageusia*) in the anterior two-thirds of the tongue from implication of the chorda tympani, and with hyper-sensitiveness to sounds (*hyperacousis*), especially to musical notes of low pitch, from paralysis of the stapedius. Cases of otitis media with implication of the Fallopian aqueduct do not, of course, show this hyperacousis.

(C) If the nerve is affected *between its emergence from the pons and the level of the geniculate ganglion*, it produces the same symptoms as in (A). Taste is unaffected. Since the diseased process in this situation almost always implicates the adjacent auditory nerve, deafness on that side is the rule. If the auditory nerve escapes, hyperacousis will occur from paralysis of the

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stapedius. Most cases of basal intra-cranial disease also present general cerebral symptoms, such as headache, giddiness and vomiting.

(D) If the nerve be attacked by disease *in the substance of the pons* it will produce facial paralysis as in (A), but taste and hearing remain unimpaired. An accompanying paralysis of the sixth nerve or nucleus is commonly present in such a lesion, owing to the fact that the facial fibres form a loop round the nucleus of the sixth nerve within the pons.

"Rheumatic" facial paralysis is generally acute in onset, the patient often wakening in the morning with complete facial paralysis. Sometimes, however, the palsy takes a day or two to develop completely. Pain in the side of the head, in the ear, eye or jaw, is frequent at the onset of the paralysis, but is probably due to the simultaneous irritation of sensory nerves by the cold which produced the facial paralysis.

In slight cases of "rheumatic" origin the paralysis may pass off completely in one or two weeks. Severe cases may last from two to eight months and may even become permanent. In severe cases which recover voluntary power, contracture of the paralysed muscles may supervene as the voluntary power returns. The mouth gradually becomes drawn back towards the paralysed side; the naso-labial fold and other furrows reappear on the weak side and may become exaggerated, so that on a casual glance at the face when at rest the healthy side seems to be the weaker of the two. But on voluntary movement there is no difficulty in observing which side has really been affected. With this contracture there is always combined a tendency to associated over-action of the affected muscles, so that on smiling or shutting the eyes there is associated contraction in the zygomatic muscles of the affected side.

The **prognosis** in facial paralysis depends upon its cause. In cases due to tumours or to caries of bone the prognosis is unfavourable. Syphilitic cases may yield to anti-specific remedies. The vast majority of cases, however, are of the rheumatic variety, and of these a large proportion recover completely. In any given case the safest basis of prognosis is founded upon a study of the electrical reactions. If, in a case of a week's duration, we find the electrical irritability of the paralysed muscles unchanged or increased or only slightly diminished, both to induction shocks and to the continuous current, we can exclude severe trophic changes and may expect recovery within three to six weeks, often within a fortnight. If a case with almost normal reactions to induction shocks

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yet shows the reactions of degeneration with the continuous current, its duration will probably be from five to eight weeks. But if the excitability of the muscles be much diminished both to induction shocks and to the continuous current, within a few days after the onset of the paralysis, a cautious prognosis should be given—three to four months or longer. The earlier we make our electrical examination after the onset of the paralysis, the more cautious should we be in prognosis. If the paralysis has only lasted two or three days it is impossible to give a prognosis from electrical examination, whilst, on the other hand, in cases examined after a week or ten days of paralysis grave errors of electro-prognosis are rare.

The **treatment** varies with the cause. In cases where there is a possibility of the disease being syphilitic, iodides and mercury should be exhibited. In ear cases the otitis should be promptly treated, surgically if necessary. In rheumatic cases a fly-blister should be applied behind and below the ear, over the point of exit of the nerve from the stylomastoid foramen; also diaphoretics and a smart purge should be administered, whilst the patient should be protected against chills. The paralysed muscles should have their nutrition maintained by electrical stimulation with a continuous current of medium intensity for about a quarter of an hour several times daily. The positive pole should be placed on some "indifferent" spot, such as the back of the neck, whilst the negative pole is stroked gently along the affected muscles.

VIII. THE AUDITORY NERVE

The eighth or auditory nerve is made up of two sets of fibres, differing in function, those of the cochlear root subserving the function of hearing, whilst those of the vestibular root go to the semicircular canals and form the nerve of equilibration.

The fibres of the latter root pass backwards internal to the restiform body to a nucleus in the floor of the fourth ventricle, whilst those of the cochlear root pass to the outer side of the restiform body and become continuous with the striæ medullares.

Centrally the nucleus of the vestibular nerve is connected indirectly with the middle lobe of the cerebellum, and with the descending antero-lateral tract of the cord. The central auditory tract connects the nucleus of the cochlear nerve with the posterior quadrigeminal body and internal geniculate body. Thence fibres pass to the cortical auditory centre at the posterior part of the upper temporo-sphenoidal convolution.

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Primary disease of the auditory nerve is rare, apart from disease in the other structures of the ear. At the base of the brain, however, the nerve may be implicated by various pathological processes, such as tumours of the bone or meninges, syphilitic disease, caries of the petrous bone, or aneurisms. In all such cases the adjacent facial nerve is commonly affected also. The nerve may also be attacked in tabes, insular sclerosis, or leukæmia.

The labyrinth may be primarily diseased, or it may become secondarily involved by extension from middle-ear disease. Labyrinthine disease may be due to inflammatory, hæmorrhagic, or sclerotic processes, or it may be syphilitic in nature. Epidemic cerebrospinal meningitis is a relatively frequent cause, by extension along the sheath of the auditory nerve, and if occurring in young children may result in deaf-mutism. Other causes are diabetes, pernicious anæmia, and leukæmia. Permanent deafness following stimulation by a loud noise is probably due to a hæmorrhage in the labyrinth. The temporary deafness which may be produced by certain drugs, such as quinine and sodium salicylate, seems also to be of labyrinthine origin.

It is often impossible to diagnose between affections of the labyrinth and those of the nerve itself. Both of them produce "nerve deafness," as tested by the absence of bone conduction of the vibrations of a tuning fork. Nerve deafness is frequently associated with subjective auditory sensations and sometimes also with giddiness and disturbance of equilibration. This latter symptom is referable to an affection either of the semicircular canals or of the vestibular root.

Symptoms of irritation or hyper-excitability of the cochlear nerve are usually referable to causes outside the nerve itself. Thus *hyperacousis*, where sounds are heard with painful intensity, which to normal ears are faint or even inaudible, may be a symptom in paralysis of the stapedial fibres of the facial nerve. More commonly, however, it is of cortical origin, occurring either in organic brain disease or, still more frequently, in hysteria. *Dysacousis*, where ordinary sounds are painful or disagreeable, is common in neurasthenia and in association with headache. *Tinnitus aurium* is a term applied to certain subjective aural sensations. The sound may be of most varied types, singing, whistling, buzzing, ticking, etc. It may be an effect of the pressure of a plug of wax against the tympanic membrane, disappearing on removal of the cerumen. More commonly it is found in anæmic and neurasthenic patients. Sometimes it occurs as an "auditory aura" in epilepsy or as part

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of an attack of migraine. The "clicking" variety of tinnitus may be due to intermittent spasm of the tensor tympani or of the levator palati. The pulsating variety of tinnitus, synchronous with the pulse, and possibly due to the close proximity of the internal carotid to the labyrinth, is commonest in neurasthenic patients, especially during the silent watches of the night. It has no pathological significance.

In every case of tinnitus the ear should be carefully examined in order to remove any source of peripheral irritation which may be detected.

MÉNIÈRE'S DISEASE.—This is a term applied to a sudden attack of tinnitus and giddiness, often followed by vomiting. The giddiness may be so severe that the patient falls to the ground as if shot. Nerve deafness may be present in the ear in which the tinnitus occurred, but is not complete as a rule. The condition is sometimes due to hæmorrhage into the vestibular nerve or semicircular canals, and also into the cochlear nerve or labyrinth in those cases where tinnitus and deafness occur. The attacks may recur at irregular intervals. Other cases of Ménière's disease are associated with pressure upon the nerve by basal growths.

IX. THE GLOSSO-PHARYNGEAL NERVE

Origin and distribution.—The nerve appears on the surface of the medulla in the groove between the olivary and restiform bodies between the facial and vagus roots. Traced inwards, the fibres are found to originate from a nucleus in the floor of the fourth ventricle, being joined by fibres from the upper part of the vagal nucleus. The nerve leaves the skull with the vagus and spinal accessory nerves by the jugular foramen.

No case of isolated disease of the ninth or glosso-pharyngeal nerve has yet been observed in man. Its precise functions are consequently less definitely known than those of any other cranial nerve. Whilst it is certain that its peripheral branches supply the posterior third of the tongue and the soft palate with taste fibres, yet it is highly probable that these taste fibres are derived from the fifth nerve, and only join the glosso-pharyngeal at the petrous ganglion. The glosso-pharyngeal is a sensory nerve for the posterior third of the tongue, the soft palate, fauces and pharynx; it is motor for the middle constrictor of the pharynx and for the stylo-pharyngeus and it is the inhibitory nerve for the movements of deglutition.

Symptoms.—Little is known about the effects of glosso-pharyngeal nerve paralysis in man. If it be affected above the petrous

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ganglion, anæsthesia of the pharynx and the back of the tongue result, along with difficulty of swallowing. In animals where the glosso-pharyngeal has been divided, the muscles of the œsophagus and pharynx remain tonically contracted from paralysis of the inhibitory fibres of the nerve. If the glosso-pharyngeal be diseased at or below the petrous ganglion, there is, in addition, loss of taste in the posterior third of the tongue on that side.

X. THE VAGUS NERVE

Origin and distribution.—The ordinary text-book descriptions of the vagus and accessorius nerves state that the vagus nerve is joined, close below its exit from the jugular foramen, by the fibres of the so-called "bulbar part" of the spinal accessory, whose roots arise from the medulla close below what was termed the vagus nerve, whilst the so-called "spinal part" of the spinal accessory, whose roots emerge from the cervical part of the spinal cord, ascends through the foramen magnum to emerge from the skull through the jugular foramen along with the vagus and so-called "bulbar part" of the accessorius.

More recent evidence, however, both physiological and clinical, goes to show that what has usually been termed the "bulbar part" of the accessorius should really be considered as belonging to the vagus itself, since it arises from a continuation of the same nucleus in the medulla (nucleus ambiguus), whilst the "spinal part" of the spinal accessory arises from cells in the cervical region of the spinal cord; moreover, the fibres of the "spinal part" of the spinal accessory nerve are large in diameter and easily to be distinguished microscopically from the small fibres of the vagus and of the so-called "bulbar part of the spinal accessory."

The spinal accessory nerve of Willis, properly so defined, and as defined originally by Willis himself, is therefore a nerve of purely spinal origin and corresponds to what, in the anatomical text-books, has been termed the "spinal part" of the spinal accessory.

On the above understanding, the fibres of origin of the vagus include, in addition to what have usually been ascribed to it, those of the so-called "bulbar part of the spinal accessory," and as such the vagus nerve will here be described. Hitherto, on the old view, it has been necessary to state that the vagus received its motor fibres for the soft palate and larynx, together with its cardio-inhibitory fibres, from the "bulbar part of the spinal accessory." But, if we regard the "bulbar part of the accessorius" as really belonging to the vagus from the first, the newer nomenclature is obviously simpler as well as more accurate.

The tenth or vagus nerve has a most extensive anatomical dis-

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tribution. It supplies the pharynx, larynx, heart, lungs, œsophagus, stomach, and even, in part, the intestines and spleen. By its auricular branch it also supplies the skin of the outer ear. Its lowest fibres of origin, as already explained, are those which are the motor fibres for the larynx and palate and the inhibitory fibres for the heart.

Its pulmonary fibres are motor for the bronchial muscles and sensory for the respiratory passages. The vagus is motor and sensory for the œsophagus, sensory for the stomach, and partly motor for the stomach and intestines.

Paralysis of the vagus nerve.—In its intra-cranial course the vagus may be implicated by various diseases of the bones or meninges and by aneurism of the vertebral artery. In the neck it may be injured by traumatism, operations, tumours or aneurisms. In the chest it may be compressed by aneurisms or mediastinal growths. Its recurrent laryngeal branch may be compressed on the right side by pleuritic apical adhesions, whilst on the left side it is specially liable to pressure by aortic aneurisms. Paralysis of the vagus may also result from poisoning by alcohol, lead, arsenic or phosphorus, or after various specific fevers, as diphtheria and influenza. It may also occur in tabes, syringomyelia and in insular sclerosis. Neuromata of the vagus itself are rare.

Symptoms.—These vary according to the site of the lesion. Intracranial lesions of the vagus may affect all its roots of origin, or may only attack its upper or lower roots. In such cases the adjacent hypoglossal nerve is frequently affected also.

If the whole vagus trunk be affected, there is unilateral paralysis of the soft palate and larynx, with anæsthesia of the larynx on that side. In cases of bilateral vagus paralysis there is tachycardia and irregularity of the cardiac rhythm from paralysis of the cardio-inhibitory fibres; there is also slowness and irregularity of respiration. Gastric symptoms have also been observed, such as vomiting, bulimia, gastric pain and loss of the sensations of hunger and thirst.

Of the above symptoms the most constant and the easiest to recognise are the affection of the soft palate and the paralysis of the recurrent laryngeal branch, which supplies all the intrinsic muscles of the larynx (except the crico-thyroid, which is innervated by the superior laryngeal branch). In *recurrent laryngeal paralysis* the affected vocal cord is seen to be immobile and in the cadaveric position, midway between abduction and adduction. The voice is generally hoarse, though not invariably so.

If *both recurrent laryngeal nerves* be affected, both vocal cords

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are motionless and in the cadaveric position. The impossibility of adduction causes total aphonia, whilst the equal impossibility of wide abduction may produce some stridor on deep inspiration.

OTHER LARYNGEAL PALSIES.—In *unilateral abductor palsy* the voice is unaffected, but on laryngoscopic examination the paralysed cord is seen to be immobile during inspiration, whilst both cords approach normally on phonation. In *bilateral abductor palsy* the voice is also unaffected and both cords approach during phonation but do not separate on inspiration. Inspiration is therefore laboured and accompanied by marked stridor. *Paralysis of the internal thyro-arytenoids*, as in early cases of bulbar paralysis, is evidenced by an oval appearance of the glottis during phonation from loss of support of these muscles. The voice is thus rendered hoarse, but abduction and adduction are otherwise unaffected.

Adductor paralysis is always bilateral, and most cases are functional in nature. There is no stridor, and during inspiration the cords appear normal. On attempted phonation, however, they do not move inwards to the slightest extent. This is the common appearance in hysterical aphonia.

XI. THE SPINAL ACCESSORY NERVE

Origin and distribution.—The eleventh or spinal accessory nerve of Willis, strictly so-called, arises from the upper cervical region of the spinal cord by a number of roots. It ascends through the foramen magnum in close apposition to the lowest fibres of the vagus (erroneously termed the "bulbar part of the accessorius," *vide supra*), and, emerging through the jugular foramen, passes backwards to be distributed to the sterno-mastoid and the upper fibres of the trapezius.

PARALYSIS OF THE SPINAL ACCESSORY NERVE.—The intracranial part of the nerve may be affected near the foramen magnum by local meningitis, tumours, or aneurisms. It may also be attacked by insular sclerosis. Outside the skull the nerve may be damaged by wounds, tumours, or abscesses. It may also be compressed by cervical caries, or involved in the cicatrix after removal of diseased "glandulæ concatenatæ," and in rare cases it is attacked by "rheumatic" neuritis.

Symptoms.—These are exclusively motor. There is weakness and wasting of the sterno-mastoid and upper fibres of the trapezius. Consequently rotation of the chin towards the opposite shoulder is defective, and there is impaired elevation of the shoulder on the side of the lesion. In cases where the nerve is involved after it has

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perforated the sterno-mastoid, this latter muscle escapes and the upper part of the trapezius is alone affected.

The electrical reaction of degenerations appear in the wasted muscles.

XII. THE HYPOGLOSSAL NERVE

Origin and distribution.—The nucleus of the twelfth nerve lies ventrally and to the side of the upper part of the central canal of the medulla, and extends upwards beneath the lower part of the floor of the fourth ventricle. Pyramidal fibres connect this nucleus, *viâ* the crus cerebri and internal capsule, with the lower part of the ascending frontal convolution. From the nucleus the fibres are directed downwards and slightly outwards, through the medulla, to reach the surface between the olivary body and anterior pyramid. Leaving the skull by the anterior condylar foramen, the nerve is finally distributed to the muscles of the tongue and depressors of the hyoid bone. Its function is purely motor.

Paralysis of the hypoglossal nerve.—The hypoglossal nerve is much more frequently affected in its intracranial course than outside the skull. Owing to their close proximity to the middle line, both hypoglossal nerves may be affected together in diseases of the medulla, and other cranial nerves are frequently affected at the same time.

Tumours and caries at the base of the skull, meningeal affections, and aneurism of the vertebral artery may affect the nerve in its intracranial course. It may also become atrophied in tabes, syringomyelia and insular sclerosis. Suboccipital caries in the region of the occipito-atlantoid joint may cause a neuritis of the adjacent hypoglossal nerve. In the neck it may be injured by wounds or compressed by tumours, but isolated hypoglossal paralysis from extra-cranial causes is very rare.

Symptoms.—There is weakness and wasting of the corresponding half of the tongue, whose mucous membrane on that side becomes markedly wrinkled. On protrusion the tongue is curved, with its concavity on the paralysed side, so that the tip deviates towards the affected side. On retraction the paralysed side of the tongue is higher than the other. The unilateral paralysis of the depressors of the hyoid bone is difficult to demonstrate. The larynx may deviate towards the sound side during the act of swallowing. The atrophied muscles show the reactions of degeneration.

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Hemiatrophy of the tongue sometimes occurs in association with facial hemiatrophy, but is probably referable to an affection of the trigeminal nerve and not of the hypoglossal. In such cases it is unaccompanied by electrical changes on the atrophied side.

Prognosis and treatment in cranial nerve palsies.—From what has been already said, it will be observed that a large proportion of cases of cranial nerve paralysis are due to intra-cranial causes, either in the form of degenerative conditions in the nerves themselves or of new growths, syphilitic or tuberculous disease or chronic inflammatory affections of the cranial bones or meninges. Many of these conditions are beyond the reach of the physician's or surgeon's art. The prognosis is best in recent cases due to syphilis, and in them the energetic administration of mercury and iodides often produces remarkable cures. It must not be forgotten, however, that when syphilitic changes have gone on to a sclerotic or fibrous stage, they are no longer amenable to anti-syphilitic medication; cure is then out of the question, although recrudescence may be prevented.

In a much smaller proportion of cases, tuberculous masses in the brain compressing various cranial nerves may be brought to a stage of quiescence or may even be absorbed, but the prognosis in tuberculous or pyogenic affections of the cranial bones or meninges is generally an unfavourable one, almost the sole exception occurring in the case of the temporal bone, where prompt surgical treatment of disease in the middle ear often relieves the facial palsy, unless the conductivity of the facial nerve has been severed by the diseased process in the Fallopian aqueduct.

It is only in a few cases that such extra-cranial causes as exposure to cold, and traumatism from stabs or bullet-wounds, come into play. The treatment of nerves secondarily involved in new growths, aneurisms, abscesses or other extra-cranial affections resolves itself into the treatment of the primary disease. In cases where a cranial nerve is accidentally divided by a stab or bullet-wound or during the course of an operation, its immediate reunion should always be attempted. During the period of waiting for regeneration to set in, the muscles supplied by the divided nerve should be kept alive by massage and electrical treatment, exactly as in the treatment of "rheumatic" facial paralysis.

When the cranial nerve paralysis is due to poisoning by lead, arsenic, mercury, etc., or by the toxins of specific fevers, such as diphtheria or influenza, similar electrical treatment should be

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carried out, and the prognosis is good unless other complications set in, *e.g.* heart-failure in diphtheria.

Degenerative conditions, such as disseminated sclerosis, tabes and syringomyelia, are incurable. The ocular palsies of early tabes, however, are often transient, passing off spontaneously.

AFFECTIONS OF THE SYMPATHETIC NERVOUS SYSTEM

Anatomy.—The sympathetic nervous system forms two gangliated cords which lie, one on each side, immediately in front of the vertebral column, extending from the base of the skull to the coccyx. They are connected above with plexuses entering the cranial cavity, whilst below they converge on the sacrum and terminate in a loop on the coccyx. Each sympathetic chain, in addition to its longitudinal association neurones, is connected with the anterior primary divisions of the spinal nerves by *rami communicantes*. The white or medullated rami communicantes are efferent neurones which pass to the sympathetic cord and usually join one of its ganglia; the gray or non-medullated rami are afferent neurones, each one passing from a multipolar cell of a sympathetic ganglion to one of the cerebro-spinal nerves.

The gangliated cord of the sympathetic gives branches, either directly or through cerebro-spinal nerves, or through the great prevertebral ganglia (cardiac, solar, and hypogastric) to the various glands and viscera of the body, to the heart and blood-vessels, to the uterus, and to the unstriped muscles of the body generally. In addition, the sympathetic innervates the dilator pupillæ, the unstriped muscle of the eyelids, the orbital muscle of Müller, also the sweat-glands and the muscles of the hair follicles. Its varied functions are thus motor, vasomotor, and secretory.

Pathology.—Traumatic lesions of the sympathetic cord occur chiefly in its cervical portion, where it may be injured by stabs, bullet-wounds, operation-wounds, or compressed by new growths, by aneurisms, or by cicatricial tissue at the apex of a tuberculous lung. It is of great importance to remember that the fibres of the sympathetic which go to the pupil and to certain muscles around the globe of the eye enter the inferior cervical ganglion of the sympathetic through the efferent rami communicantes of the first thoracic nerve root. Hence symptoms of cervical sympathetic paralysis may also be caused by lesions of the spinal cord in the cervical region, either acute, as in traumatism, or chronic, as in syringomyelia.

Lesions of the thoracic or abdominal portion of the sympathetic are less frequently observed. The implication of the solar plexus

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of the sympathetic in some cases of Addison's disease has already been referred to (Vol. II. p. 113). The sympathetic chain may also be compressed or infiltrated by tumours, abscesses, etc., in adjoining structures.

Symptoms.—In *paralysis of the cervical sympathetic* there is narrowing of the pupil on the affected side, from paralysis of the dilator pupillæ. Moreover, the pupil neither dilates to shade nor to the instillation of cocaine, although it still contracts on exposure to light. The cilio-spinal reflex:—dilatation of the pupil on pinching the skin of the neck—is also lost on the affected side. The palpebral fissure becomes narrowed—a form of pseudo-ptosis referable to paralysis of the non-striped muscle of the eyelid, since the upper eyelid can still be voluntarily elevated to its full extent by the levator palpebræ. The globe of the eye also sinks back, this *enophthalmos* being ascribed to paralysis of the orbital muscle of Müller, and sometimes the tension of the globe is diminished. The cutaneous vessels of the corresponding side of the face and scalp are sometimes dilated, from vasomotor paralysis; this, however, often passes off, and is succeeded by a secondary and permanent contracture of the vessels. *Anidrosis* or absence of sweat sometimes occurs on the affected side, being sharply limited by the middle line. In one case of bullet-wound observed by the writer, implicating the cervical sympathetic, the area of anidrosis extended to the level of the third thoracic spine behind, to the third rib in front, and included the whole of the upper extremity on the affected side. Slight flattening of one cheek has been observed in a few cases. No constant alteration in cardiac ryhthm has been noticed in such unilateral cases, possibly because the cardiac nerves of the other side suffice to regulate the heart.

Cases of unilateral *irritation of the cervical sympathetic* are rare, and most of them go on to paralytic lesions. The symptoms of irritation of the cervical sympathetic are the converse of those produced by its paralysis, viz. dilatation of the pupil, widening of the palpebral fissure, forward projection of the eyeball (*exophthalmos*), and delayed descent of the upper lid during downward rotation of the eye (von Graefe's sign). All these oculo-pupillary changes may be produced in the normal eye by the instillation of cocaine, which stimulates the cervical sympathetic.

Certain of the symptoms of exophthalmic goitre, notably the exophthalmos, which is sometimes unilateral, and the tachycardia, may be explained as possibly due to stimulation of the sympathetic (see Vol. II. p. 153); whilst the gastro-intestinal disturbances and

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the occasional pigmentary and other changes in the skin which occasionally occur in that disease may be, in part at least, explained as the result of stimulation of the sympathetic by some toxin, perhaps derived from the thyroid gland. This point, however, is much disputed.

There is also a good deal to be said in favour of the probability of such angio-neurotic conditions as acute circumscribed œdema, hydrops articulorum intermittens, erythromelalgia, and Raynaud's disease, as being largely dependent on affection of the sympathetic nerves, but opportunities for the pathological investigation of such cases are rare, inasmuch as they are not of themselves fatal.

AFFECTIONS OF THE SPINAL NERVES

THE PHRENIC NERVE

Origin and distribution.—The phrenic nerve is mainly derived from the fourth cervical root, with additional fibres from the third or fifth, and is distributed to the diaphragm, also giving filaments to the pleura, pericardium, inferior vena cava, and right auricle.

Paralysis of the phrenic nerve.—Its paralysis is usually due to some affection of the cervical vertebræ or meninges, as in fracture or spinal hæmorrhage, or in more chronic diseases of the cord, inflammatory, syphilitic, tuberculous, or in tumours. Owing to its deep position in the neck the phrenic nerve is rarely injured by wounds, but it may be compressed by tumours and aneurisms in the neck or thorax, or involved in inflammatory affections of the pleura. "Rheumatic" and toxic neuritis also occurs, and bilateral paralysis may occur in alcoholic paralysis, beri-beri, lead poisoning, and post-diphtheritic paralysis. Paralysis of the diaphragm may also result from myositis by direct extension of inflammation from an adjacent pleurisy or peritonitis.

Symptoms.—The effect of paralysis of the phrenic nerve is inaction of the corresponding half of the diaphragm. Unilateral paralysis can only be recognised by careful comparison of the movements on the two sides. Bilateral affection of the phrenic nerves causes total paralysis of the diaphragm, which no longer contracts on inspiration. On the contrary, in such a case, instead of the normal advance of the epigastrium during inspiration we find

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actual recession. The movements of the upper part of the thorax are excessive, to compensate for inaction of the diaphragm, but dyspnœa is not necessarily present when the patient is at rest, although it supervenes on the slightest exertion. Moreover, any lung disease, such as bronchitis or pneumonia, is rendered much more serious by the respiratory inefficiency.

The **prognosis** is good in rheumatic and in post-diphtheritic cases, less so in alcoholism, and worst in central disease of any kind.

Treatment.—Electrical stimulation of the paralysed nerve is advisable only in cases of peripheral origin. In cases of diaphragmatic palsy due to lesions in the cord, treatment by electricity is not indicated; attention should then be devoted to the central lesion.

The nerve can be stimulated by placing one pole of the battery deeply to the outer side of the clavicular head of the sterno-mastoid, the other pole being placed over the epigastrium.

Hypodermic injections of strychnia are of benefit in alcoholic and post-diphtheritic cases.

THE BRACHIAL PLEXUS

Origin and distribution.—The brachial plexus is derived from the anterior primary divisions of the four lowest cervical nerves and of the first dorsal nerve. Of these, the anterior primary divisions of the fifth and sixth cervical nerves unite to form the "upper trunk" of the plexus. The seventh cervical runs on as the "middle trunk," whilst the eighth cervical and first dorsal unite to form the "lower trunk." Each of these trunks divide into an anterior and a posterior branch, of which all three posterior branches unite to form the "posterior cord" of the plexus, the anterior branches of the upper and middle trunks combining to form the "outer cord"; whilst the "inner cord" is formed by the anterior branch of the lower trunk. The cords of the plexus closely embrace the axillary artery. For a further description of the origin and distribution of the various supra- and infra-clavicular branches of the plexus, the reader is referred to anatomical text-books.

Paralysis of the brachial plexus.—The plexus may be paralysed in whole or in part. It is not always possible to tell whether the site of the lesion is in the plexus itself or in the nerve roots before they form the plexus. Moreover, a lesion often extends from the roots into the plexus.

Paralysis of the whole brachial plexus is rare. It is almost always traumatic, resulting from injuries in the region of the

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shoulder. Thus it may occur during a dislocation, where the head of the humerus compresses or ruptures the plexus, as in some birth palsies. Fracture of the clavicle, or callus-formation after a fracture, may also cause plexus paralysis. Stabs and blows in the region of the shoulder, and forcible approximation of the clavicle to the first rib, may affect either the plexus or the roots of which it is constituted. Tumours in the supra-clavicular region and subclavian aneurisms may also compress the plexus.

The **symptoms** are those of flaccid atrophic paralysis of the whole of the muscles of the upper limb. Sensory affection is generally present, but varies in extent in different cases and is not always proportional to the motor palsy.

Recovery, as a rule, except in cases of temporary compression, is incomplete, certain muscles remaining permanently atrophied.

Two varieties of partial plexus palsy merit special description, the one type affecting the two highest, the other, a less frequent variety, affecting the two lowest, roots of the plexus:—

ERB-DUCHENNE PARALYSIS is a palsy affecting the deltoid, biceps, brachialis anticus, and supinator longus muscles. In addition, the supinator brevis is frequently affected, sometimes also the infra-spinatus, more rarely the subscapularis. The lesion is situated in the fifth and sixth cervical roots, either above the brachial plexus or after they have united to form the "upper trunk." The causes are those already described for total plexus paralysis.

Symptoms.—The affected muscles are atrophied, and exhibit the electrical reactions of degeneration. Deltoid paralysis is evidenced by inability to abduct the arm at the shoulder-joint, whilst the affection of the biceps, brachialis anticus, and supinator longus is shown by inability to flex the elbow-joint, which hangs loosely extended. Supination of the forearm is still possible, unless the supinator brevis be paralysed, in which case the hand is persistently in a pronated posture. If the infra-spinatus be affected the shoulder-joint is habitually rotated somewhat inwards and cannot be voluntarily rotated outwards, hence the act of writing is rendered difficult. If the subscapularis be paralysed, inward rotation of the shoulder-joint is impaired, and when this muscle becomes atrophied passive movements of the scapula cause a grating noise from the rubbing of the unpadded scapula against the ribs. Sensation may be impaired in the cutaneous distribution of the fifth and sixth cervical segments along the outer aspect of the upper limb, from the middle of the deltoid down to the thumb, index, and adjacent

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side of middle finger (*vide* Table of Spinal Localisation, p. 210), but in some cases sensation is unaffected, or more probably has recovered before the case comes under observation.

Klumpke's paralysis is due to an affection of the lower roots of the brachial plexus. The lesion is in the eighth cervical and first dorsal roots, either separately above the plexus or after they have united to form the "lower trunk." The causes are the same as for total plexus palsy.

Symptoms.—The muscles which atrophy in this variety of paralysis are the intrinsic muscles of the hand and the flexors of the fingers and wrist. Paralysis of the interossei produces inability to spread out or to approximate the fingers, or to extend the interphalangeal joints. The thenar and hypothenar eminences become flattened, and opposition or adduction of the thumb becomes impossible. Paralysis of the flexors of the fingers and wrist renders the patient unable to bend those joints or to grasp anything in his hand.

If the nerve roots are affected closer to the cord than the point where the rami communicantes are given off to the cervical sympathetic, oculo-pupillary changes on that side are present, consisting in narrowing of the palpebral aperture and sinking in of the eyeball (from paralysis of Müller's muscle), with contraction of the pupil and absence of dilation on shading the eye or on instilling cocaine. The cilio-spinal reflex is also abolished on the side of the lesion.

It should be remembered that a paralysis which originally affects the whole brachial plexus may ultimately clear up either to an Erb-Duchenne or to a Klumpke paralysis.

Paralysis of Individual Nerves of the Upper Extremity

Causation.—The individual nerves are liable to paralysis from various causes, of which by far the most frequent is traumatism. In fractures and dislocations the adjacent nerves may actually be torn, or they may be compressed, either by the fragments themselves or by callus formed later. Thus fractures of the shaft of the humerus tend to implicate the musculo-spiral nerve; fractures of the bones of the forearm often affect the median, whilst fractures and dislocations in the region of the elbow-joint are specially likely to cause ulnar paralysis. "Sleep palsies" are not uncommon, especially when the patient is under the influence of alcohol or other narcotics, the median and musculo-spiral nerves being those most commonly so

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compressed. "Crutch paralysis" affects specially the circumflex nerve or the musculo-spiral. Stabs, bullet wounds, blows of various kinds, and tourniquets around the limb may injure any of the nerve trunks. The posterior thoracic and supra-scapular nerves may be injured by carrying heavy weights on the shoulder. Powerful muscular contraction of the triceps has caused musculo-spiral paralysis. Constant muscular contraction of the scalenus, as in plasterers and others who have the arm continually raised at work, may set up neuritis in the posterior thoracic nerve, which perforates the scalenus medius.

Primary neuritis of individual nerves is less common, but may occur after various infective fevers, such as diphtheria, influenza, and septicæmia, also after rheumatic fever, in diabetes, and in various other conditions.

PARALYSIS OF THE POSTERIOR THORACIC NERVE

Symptoms.—In such a case the only muscle affected is the serratus magnus. When the arm is at rest, the scapula on the affected side is displaced slightly upwards by the levator anguli scapulæ and rhomboids, and its lower angle is tilted towards the middle line. On abduction of the humerus to the horizontal line, the inner border of the scapula comes still farther inwards and projects backwards from the trunk. On holding the arm horizontally forwards, this "winging" of the scapula becomes still more evident, and between its inner border and the chest-wall a gap appears, into which the observer's hand can be placed. The patient usually has difficulty in elevating his arm above the horizontal line, since the necessary outward movement of the scapula produced by the serratus is deficient; but if the observer pushes the angle of the scapula outwards, the patient is then able to raise the arm easily to the vertical position.

PARALYSIS OF THE CIRCUMFLEX NERVE

The chief **symptom** is paralysis of the deltoid. Complete abduction of the arm to the horizontal line is impossible, though the supra-spinatus may raise the arm to a slight extent. The associated paralysis of the teres minor is clinically difficult to recognise. Sometimes there is anæsthesia of the skin over the deltoid muscle, which is supplied by the circumflex nerve. Adhesions in the shoulder joint are apt to form, probably due in part to trophic changes.

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PARALYSIS OF THE MUSCULO-SPIRAL NERVE

Owing to its peculiar course and superficial position, this nerve is more commonly injured than any other nerve of the upper extremity. The **symptoms** depend upon the level of the lesion. In a complete case we find paralysis of the extensors of the elbow and wrist, of the supinators and of the long extensors of the thumb and fingers. In most cases, however, the nerve is affected after giving off its branches to the triceps. Paralysis of the triceps is shown by inability to extend the elbow against gravity or against resistance, although the joint hangs extended by the weight of the forearm. Flexion of the elbow is slightly weakened from affection of the supinator longus, and the latter muscle does not stand out on flexing the elbow against resistance. Paralysis of the supinator brevis renders supination impossible with the elbow extended, although when the elbow is in the flexed position the biceps can still supinate powerfully. From paralysis of the extensors there is wrist-drop and the fingers and thumb are slightly flexed, the thumb being also opposed. Neither the wrist nor the metacarpo-phalangeal joints can be extended, although the interphalangeal joints can still be extended by the healthy interossei.

Flexion of the wrist and fingers is still possible, but the grasp is feeble, owing to the abnormal position of the hand. But if the wrist be passively hyper-extended the grasp is very much less impaired, since the flexors can then act at a greater advantage.

In cases due to pressure, sensation is but little affected, but when the nerve is torn across or divided, there is anæsthesia of the radial side of the dorsum of the hand and of the thumb, index, middle, and part of the ring finger, supplied by the radial nerve, and if the lesion be high up there is also anæsthesia of the area on the outer side of the upper arm and on the back of the forearm, supplied respectively by the external and internal cutaneous branches of the musculo-spiral.

PARALYSIS OF THE MEDIAN NERVE

Distribution.—The median nerve supplies the two pronators, the flexor carpi radialis, the flexor longus pollicis, the flexor sublimis and the radial half of the flexor profundus digitorum. In the hand it supplies the abductor and opponens pollicis, part of the flexor brevis pollicis and the two radial lumbricales. It is also sensory for the palmar aspect of the thumb, index, middle, and half of the ring finger,

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with the corresponding part of the palm of the hand, and for the dorsum of the terminal phalanx of each of those digits.

Symptoms.—Median paralysis is relatively rare. When the palsy is complete, the forearm is slightly supinated, owing to paralysis of the pronators. Pronation is impossible, and the patient endeavours to compensate for this by rotating the shoulder inwards. The hand is drawn slightly to the ulnar side by the flexor carpi ulnaris. Flexion of the wrist is feeble and during the act there is strong deviation to the ulnar side. Flexion of all the proximal interphalangeal joints is lost and of the distal interphalangeal joints of the index and middle finger. The metacarpo-phalangeal joints can still be flexed by the interossei. In long-standing cases the unopposed action of the interossei on the interphalangeal joints tends to produce in them a backward dislocation. The thumb is persistently extended and adducted to the index, producing an "ape-like" hand. Flexion of the terminal joint of the thumb is impossible, and the thumb cannot be opposed to the little finger. Vasomotor and trophic changes may occur in the skin and nails of the affected fingers, and there is cutaneous anæsthesia in the area above described.

PARALYSIS OF THE ULNAR NERVE

Distribution.—The ulnar nerve supplies the flexor carpi ulnaris, the ulnar half of the flexor profundus digitorum, the adductor policis and deep head of the flexor brevis pollicis, together with all the interossei, the hypothenar muscles and the two ulnar lumbricales. It is also the sensory nerve for the ulnar side of the hand, the little finger, and ulnar half of the ring finger, both on palmar and dorsal aspects.

Symptoms.—In ulnar paralysis flexion of the wrist is still possible, but during flexion the hand deviates to the radial side. The patient cannot flex the distal interphalangeal joints of either the little or ring finger, although flexion of the proximal interphalangeal joints of all the fingers and of the distal interphalangeal joints of the middle and index finger is still possible.

The most marked symptoms, however, are those produced by the paralysis of interossei and lumbricales. Flexion of the metacarpo-phalangeal joints is impossible and extension of the interphalangeal joints is also lost. The two ulnar fingers are more affected in this respect than the other two fingers, owing to the fact that the median nerve supplies two of the lumbricales. Spreading out and approximation of the fingers is impossible.

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In old-standing cases contracture of the extensors of the fingers produces a "claw-hand," this being most marked in the two ulnar fingers for the same reason. The hypothenar eminence becomes wasted and the palm is hollowed.

Occasionally trophic changes occur in the skin of the affected fingers. Anæsthesia affects the hand in the area above described, unless the nerve is injured close above the wrist, when its dorsal cutaneous branch may escape.

PARALYSIS OF THE LUMBAR AND SACRAL PLEXUSES AND OF THE INDIVIDUAL NERVES OF THE LOWER EXTREMITY

Origin and distribution.—The lumbar plexus is formed by the anterior primary divisions of the first, second, third, and half of the fourth lumbar nerves. The nerves entering the lumbar plexus do not form an interlacement, as in the brachial plexus, but the several nerves of distribution proceeding from the plexus for the most part arise by two or more roots from a corresponding number of spinal nerves, so as to produce the appearance of a series of loops.

The sacral plexus is formed by half of the fourth lumbar, the fifth lumbar (these constituting the lumbo-sacral cord), and the first four sacral nerves. There is but little interlacement of these nerves.

Causation.—The plexuses and nerves of the lower extremity are affected much less frequently than the brachial plexus and the individual nerves of the upper limb.

The lumbar plexus may be damaged by tumours of the vertebræ or of the retro-peritoneal lumbar glands, or by psoas abscess. The anterior crural nerve itself may be injured in fractures of the pelvis or in dislocations of the hip, also by stabs or bullet-wounds in the groin. The obturator nerve is rarely paralysed alone, but may be compressed by a pelvic tumour or an obturator hernia. The anterior crural and obturator nerves are both liable to be compressed during parturition. Spontaneous neuritis occasionally occurs in the lumbar plexus or its branches, as in gout, diabetes, or alcoholism.

The sacral plexus may be compressed by tumours of the lumbar or sacral vertebræ, and by intra-pelvic growths or exudations. It may also be injured during parturition, sometimes when forceps have been used, but also in cases where there has been no such instrumental interference. The chief branches of the sacral plexus are the great and small sciatic nerves and the gluteal nerves. Neuritis may arise spontaneously in the plexus; more commonly, however, it is an extension from the sciatic (*vide* "Sciatica," p. 281).

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The sciatic nerve itself may be injured in dislocations of the hip, in fractures of the femur, and by bullet-wounds or stabs. Whether the lesion be in the sacral plexus or in the sciatic nerve itself, the symptoms in the majority of cases are most marked in the external popliteal branch of the sciatic. Moreover, the external popliteal, from its anatomical course, is more exposed to traumatic influences than the internal popliteal.

PARALYSIS OF THE ANTERIOR CRURAL NERVE

Symptoms.—Of these the chief is paralysis and wasting of the quadriceps extensor, with inability to extend the knee and loss of the knee-jerk. Owing to paralysis of the sartorius and pectineus, flexion of the hip is also weakened, and this is still more marked if the lesion be high up within the pelvis, so as to involve the branch to the iliacus. Walking is rendered difficult, since the patient cannot flex the hip properly and also because he must avoid flexing the knee, since he cannot voluntarily extend it again. The patient cannot rise from the kneeling posture without using his hands.

Sensation is impaired over the cutaneous areas supplied by the middle and internal cutaneous nerves, and by the long saphenous, viz. along the anterior and inner surfaces of the thigh in its lower two-thirds, and along the inner margin of the leg and inner border of the foot as far as the great toe.

PARALYSIS OF THE OBTURATOR NERVE

Symptoms.—Paralysis and wasting of the adductor muscles of the thigh is produced, so that the patient cannot put one knee across the other. Riding is also rendered difficult. There is anæsthesia of the upper third of the inner surface of the thigh.

PARALYSIS OF THE SCIATIC NERVE

This is seldom complete. The **symptoms** vary according to the level at which the nerve is injured. When the lesion is high up, close to the sciatic notch, there is paralysis and atrophy of the biceps, semi-membranosus and semi-tendinosus, in addition to paralysis of the external and internal popliteal nerves which supply all the muscles below the knee. The hamstring muscles flex the knee and also extend the hip, as in ordinary progression, and

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when they are paralysed walking is much impaired. The patient raises his thigh excessively high, so that the weight of the leg may flex the knee.

PARALYSIS OF THE GLUTEAL NERVES

Distribution.—The superior gluteal nerve emerges from the pelvis through the great sacro-sciatic foramen above the pyriformis muscle, whilst the inferior gluteal nerve emerges below the pyriformis. The superior gluteal nerve supplies the gluteus medius, gluteus minimus, and tensor fasciæ femoris. The inferior gluteal nerve supplies the gluteus maximus.

Causes and symptoms.—This rare paralysis may result from diseases of the sacrum and pelvis, or from fractures, stabs, or bullet-wounds in the vicinity. The symptoms are purely motor, and consist in paralysis and atrophy of the gluteal muscles together with the pyriformis and tensor fasciæ femoris. Paralysis of the gluteus maximus does not prevent the patient from standing or from walking on level ground, since the weight of the limb serves to extend the hip. But forcible extension, when the joint is previously flexed, as in going upstairs or up-hill, or in rising from a seat, is impaired. Paralysis of the gluteus medius and minimus causes loss of abduction and circumduction of the hip, hence walking becomes clumsy. The tensor fasciæ femoris rotates the thigh inwards and slightly helps in flexing the hip. When it is paralysed, the foot tends to turn outwards when it is swung forwards in the act of walking.

PARALYSIS OF THE EXTERNAL POPLITEAL NERVE

Distribution.—The peroneal nerve supplies the tibialis anticus, extensor proprius hallucis, extensor longus digitorum and all three peronei muscles. It is also sensory for the outer side of the leg and, through its musculo-cutaneous and anterior tibial branches, for the dorsum of the foot and toes.

The **symptoms** produced by its paralysis are loss of power of dorsi-flexing or everting the ankle or of extending the toes. The foot drops from its own weight and the hip has to be flexed excessively in walking to prevent the toes from catching on the ground. In long-standing cases, if the internal popliteal nerve be unaffected, contracture of the opposing calf muscles may fix the foot in a position of talipes equinus, and contracture of the

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unopposed interossei may cause persistent flexion of the metatarso-phalangeal joints.

Anæsthesia over its cutaneous distribution also occurs, and there may also be vasomotor and trophic changes in the skin.

PARALYSIS OF THE INTERNAL POPLITEAL NERVE

Distribution.—This nerve supplies the popliteus, the calf muscles, tibialis posticus, flexor longus digitorum, flexor longus hallucis, and all the intrinsic muscles of the sole. It is also sensory for the postero-external surface of the leg, and for the outer border and sole of the foot.

Symptoms.—Paralysis of the popliteus causes difficulty of inward rotation of the flexed knee. The affection of the calf muscles produces inability to extend the ankle or to stand on tiptoe. The ankle cannot be inverted nor can the toes be flexed.

If the external popliteal group of muscles be unaffected, talipes calcaneo-valgus may result from contracture, and the interosseal paralysis may produce claw-foot by a mechanism similar to that causing claw-hand.

Anæsthesia and trophic changes may occur in the area of cutaneous distribution of the nerve.

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MULTIPLE NEURITIS

Multiple neuritis, as its name implies, is a disease in which many nerves are affected, either simultaneously or successively. The condition is usually symmetrical, affecting the nerves equally on both sides; and the changes are most intense at the periphery of the nerves, lessening gradually towards their central ends.

Pathology.—The pathological alteration is a two-fold one, both degenerative and inflammatory changes being present. The degenerative change consists in atrophy of the axis cylinders and breaking

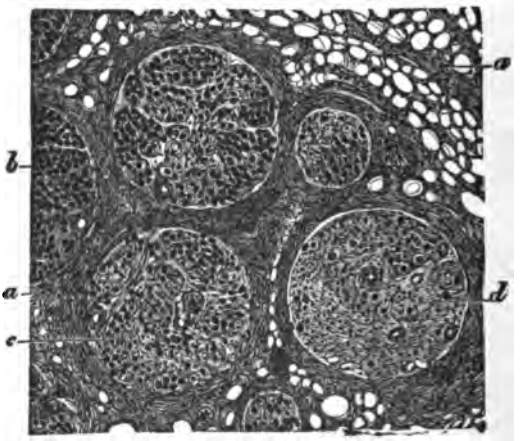

FIG. 26.—Transverse section of a peripheral nerve showing interstitial and degenerative changes. *a*, epineurium; *b*, transverse section of a normal nerve bundle; *c*, *d*, tranverse section of atrophic nerve bundles.—From Zeigler's *Pathological Anatomy*.

up of the medullary sheaths into irregular globules—so-called "parenchymatous" neuritis. The inflammatory change—so-called "interstitial" neuritis—consists in a proliferation of the fibrous tissue of the nerve sheaths. In many cases the interstitial changes are absent. When present, they are usually secondary and much less marked than the degenerative changes. Atrophy, on the other hand, is almost constantly present, more or less complete. Cases in which the interstitial changes are primary and preponderating, as

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in gouty neuritis, are usually irregular in distribution and due in part to some local exciting cause at that point. Within recent years changes in the motor cells of the anterior cornua of the spinal cord in cases of multiple neuritis have been demonstrated by Nissl's method, corroborating the general rule that, in injury to any part of a neurone, the whole element suffers. The symmetry of the degenerative changes is due to the fact that the cause is one which affects the whole nervous system and attacks most severely those parts of it which are most remote from the trophic centres, that is to say, the periphery of the nerves. The extent of the pathological changes is proportional to the severity of the clinical phenomena.

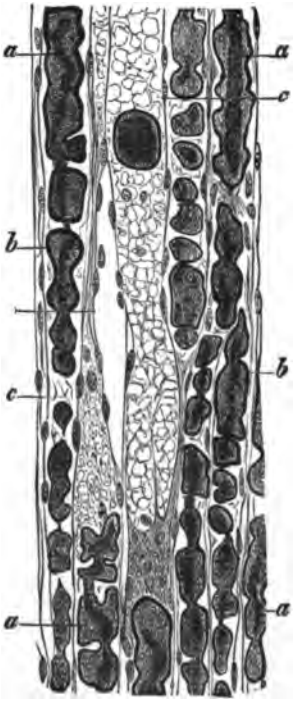

FIG 27. — Degeneration of nerve — longitudinal section *a*, remains of the axis cylinder; *b*, disintegrated medullary sheath; *c*, sheath of Schwann. — From Zeigler's *Pathological Anatomy*.

Etiology.—The causes of multiple neuritis may be classified provisionally as follows :—

i. *Cases due to non-microbic poisons :*
   (1) Metallic poisons, *e.g.* lead, arsenic.
   (2) Non-metallic poisons, *e.g.* alcohol, phosphorus, diabetes, rheumatism, gout.

ii. *Cases due to microbic poisons :*
   (1) Primary forms of neuritis, where neuritis is an early effect, *e.g.* beri-beri.
   (2) Secondary forms of neuritis, in which the neuritis is an occasional sequel of another disease, *e.g.* diphtheria, septicæmia (including puerperal cases), smallpox, malaria, influenza, enteric fever, tubercle, syphilis, etc.

iii. *Cachectic and senile cases*, due to general mal-nutrition, the vitality of the nervous system being lowest at the periphery.

Some forms of neuritis are endemic, *e.g.* malarial neuritis and beri-beri. Beri-beri may be epidemic.

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Alcoholic Neuritis

Alcoholic neuritis is commoner than all the other varieties put together. It may therefore be taken as the type, and the other forms of neuritis may be studied with reference to the points in which they differ from the alcoholic variety.

This is a disease of adult life. It affects the female sex three times as frequently as the male. It does not follow a mere occasional bout, but results from the habitual drinking, usually, of strong spirits for a period extending over several months at least. The attack of neuritis is frequently precipitated by some accessory cause, such as exposure to cold or insufficient food.

Symptoms.—The onset is generally acute or sub-acute. The earliest symptoms, as a rule, are sensory and slight in degree. They consist in numbness and tingling of the extremities, varying in intensity in different cases. Dull pains in the limbs are common, and these are made worse by movement of the limb or by pressure. Sometimes the skin is tender to the touch, while the nerve trunks and especially the muscles are always abnormally tender on pressure. Later, cutaneous anæsthesia may develop and is distributed symmetrically in the periphery of the limbs. As the disease advances, the anæsthesia gradually extends upwards on the affected limbs. This combination of anæsthesia to touch along with hyperæsthesia to pain is almost pathognomonic of the disease. The hyperæsthesia is especially marked on the soles of the feet, and may be so intense and persistent as to prevent the patient from walking, long after she has recovered from the motor weakness.

To these sensory symptoms motor weakness is soon superadded, especially affecting the legs. The gait assumes a shambling character from weakness of the peronei and anterior tibial muscles. There is double foot-drop, and the patient lifts her feet abnormally high in order to get the toes to clear the ground when walking. In severe cases double wrist-drop may also develop, from paralysis of the posterior interosseous group of muscles. This weakness of limbs may increase until the patient becomes totally unable to walk or to feed herself.

The feet are affected earlier and more frequently than the hands, and as a rule more severely. The upper extremities often escape, or may exhibit, as their only abnormality, slight anæsthesia of the finger-tips. It is rare for the disease to begin in the hands. Some cases show unsteadiness and ataxy of the limbs more pro-

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minently than actual weakness. Such cases have been termed "alcoholic pseudo-tabes," but they are rare.

The affected muscles have the electrical "reactions of degeneration," and these electrical changes may be quite distinct even in some of the non-paralysed muscles.

The knee-jerks and other deep reflexes are almost invariably lost or diminished. In rare instances they may be increased, but only in slight cases and in the early stage of the disease. The skin reflexes are generally diminished or absent, except in hyperæsthesia of the soles, when there may be an excessive response in the non-paralysed muscles. The bladder and rectum are rarely affected, as contrasted with paralysis from spinal cord disease. In rare cases incontinence or retention of urine may supervene, but this is usually from psychical dulness or in the course of delirium tremens, and therefore not referable directly to the neuritis. Amenorrhœa is common.

Trophic changes are most marked in the muscles. They become soft, flabby and wasted, although the muscular atrophy may occasionally be masked by œdema or by excess of subcutaneous fat. In the skin there is frequently excessive sweating of the feet and hands, whilst œdema is not uncommon, especially in the feet. The skin of the feet is often red and abnormally warm, and sometimes "glossy skin" develops. The joints of the feet and ankles tend to become fixed by adhesions and the unopposed calf muscles may undergo obstinate contracture. Bed-sores, however, are excessively rare.

The cranial nerves are rarely affected. The pupils remain normal, but nystagmus on extreme lateral deviation is not uncommon. In very severe cases the phrenic nerves may become affected, causing paralysis of the diaphragm, and the vagi also, with rapidity of pulse and affection of respiration.

In addition to the above symptoms referable to the neuritis, we commonly find the concomitant signs of alcoholism. Psychically we note a "habitual inaccuracy" of statement. Most patients repudiate with indignation any suggestion of alcoholism. The memory is impaired and sometimes the patient has babbling delirium with hallucinations and illusions of various kinds. The gastric troubles and the tremors characteristic of chronic alcoholism are frequently present also.

The annexed Table represents in a convenient form the main differences in the characters of the several varieties of muscular atrophy—spinal, neuritic, and myopathic.

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Muscular Atrophy

	Spinal.	Neuritic.	Myopathic.
	From chronic disease in cells of anterior cornua — Progressive Muscular Atrophy and Amyotrophic Lateral Sclerosis.	From disease in peripheral nerves—Multiple Neuritis.	So-called Primary or Idiopathic Muscular Dystrophies—Pseudo-hypertrophic Myopathy and Idiopathic Muscular Atrophy.
Muscles earliest affected.	Most commonly atrophy begins in small muscles of hand; spreads upwards to muscles whose spinal nuclei are in same or neighbouring segment of cord. Triceps, latissimus dorsi and sternal head of pectoralis major, usually the last to be affected. If trapezius is involved, its uppermost fibres rarely atrophy.	Chiefly anterior tibial group of muscles in legs, and extensors of wrists and fingers in forearms.	In pseudo-hypertrophic paralysis, condition usually begins with enlargement and hardness of calf muscles, then of quadriceps and glutei. Anterior tibial and hamstring muscles usually atrophy without previous enlargement. In upper limbs small muscles of hand often escape; certain muscles become enlarged:—deltoid, supra- and infra-spinatus and triceps. Others waste from the first, especially sternal head of pectoral, latissimus dorsi and biceps. In idiopathic muscular atrophy condition often begins in face. When arms are affected, biceps, brachialis anticus, triceps and supinator longus atrophy early. Small muscles of hand and forearm muscles, except supinator longus, usually escape. May have trapezius completely atrophied, upper as well as lower fibres. In lower limbs glutei, hamstrings, and quadriceps chiefly affected. Muscles below knee less frequently atrophy.
Age of onset.	Usually after middle life.	Usually adults.	Pseudo-hypertrophic — usually under ten years of age. Idiopathic muscular atrophy—usually between ages of fifteen and forty.
Sex.	Males oftener than females.	Females in vast majority of cases.	Pseudo-hypertrophic — affects males almost exclusively. Idiopathic — sexes almost equally affected.
Hereditary transmission.	Not hereditary.	Not hereditary.	Hereditary — Pseudo-hypertrophic transmitted through females only.

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MUSCULAR ATROPHY—*continued*

	SPINAL.	NEURITIC.	MYOPATHIC.
Electrical reactions of affected muscles.	"Partial" reaction of degeneration. Reaction to faradism diminished according to number of muscular fibres affected. Reaction to galvanism similarly diminished. No qualitative "polar" changes.	"Reaction of Degeneration." To faradism, no reaction. To galvanism, sluggish reaction with reversed polar reactions.	Electrical reactions normal to faradism and galvanism, so long as any fibres of the affected muscles survive.
Fibrillary twitchings.	Present	Absent.	Absent.
Sensory changes.	None.	Hyperæsthesia of muscles on pressure. Cutaneous anæsthesia and hyperalgesia.	None.
Knee-jerks.	Increased in amyotrophic lateral sclerosis; usually normal in progressive muscular atrophy, unless atrophy involves quadriceps, when the knee-jerk is diminished or absent.	Knee-jerks absent.	Knee-jerks never increased; may become diminished or lost, if quadriceps involved.

In fatal cases death usually occurs from failure of respiration where the intercostal nerves become affected in addition to the phrenics. Bronchitis and heart failure often hasten the end, but any acute disease which supervenes in such patients may prove fatal.

Prognosis.—Most cases recover completely, though slowly. Months or years may elapse before the patient completely recovers. In any particular case the prognosis should be guided by the

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motor rather than by the sensory symptoms. If the arms, and especially if the trunk muscles be affected, recovery is much slower than where the legs alone are weak. The disease reaches its maximum in several weeks or months. It then remains stationary for a considerable period, which may last one or two months before improvement sets in. The nerves which were attacked latest are the earliest to recover. As might be expected, recurrences of the disease are not uncommon.

Treatment.—The first object in treating alcoholic neuritis is to remove the cause. Alcohol must be cut off absolutely and at once. A trustworthy trained nurse should be obtained, or, if that be impossible, the patient should be transferred to a hospital, since such patients, if treated in their own homes, often succeed in obtaining secret supplies of alcohol. If cardiac weakness requires the administration of some stimulant, it should, if possible, be a non-alcoholic one, such as carbonate of ammonia or digitalis.

The patient should be kept at rest in bed for several weeks at least. If the pains are severe, they may be relieved by local hot applications or by antipyrin or caffein internally. When these fail, morphia may be administered hypodermically, with caution. For the alcoholic craving cocaine may be given internally in small doses or a mixture containing strychnia and capsicum. Strychnia should also be given in moderate doses by hypodermic intra-muscular injection. Electricity should be employed from the outset to maintain the nutrition of the muscles, and, later, massage and passive movements should be added, but not until the acutely painful stage of the disease is past. The feet should be protected from the pressure of the bedclothes by means of a cage, and care must be taken to prevent contracture of the calf muscles. It may be necessary to apply special splints for that purpose.

Throughout the disease tonics and careful feeding should be administered.

Diabetic Neuritis

This form of multiple neuritis presents similar symptoms of symmetrical atrophic paralysis with neuralgic pains, but without the causal antecedent of alcoholism and with the presence of sugar (not necessarily large in amount) in the urine. It should be remembered that many cases of diabetes mellitus show absence of the knee-jerks, even when there is no obvious motor weakness of the legs. As to therapeutics, the diabetes should be treated,

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dietetically and otherwise, whilst electricity, massage, and passive movements should be applied to the affected muscles. Under such treatment the paralysis may recover, but is apt to recur.

Multiple Neuritis from Lead Poisoning

This is a peculiar form of paralysis tending to affect special nerves, but almost never implicating their sensory fibres. Certain occupations involve a liability to lead poisoning. Lead-workers, especially those who make white lead, are most commonly affected, but it is frequently found also in plumbers, painters, compositors, type-founders, and pottery-glazers. Apart from working in lead, numerous accidental sources of lead poisoning exist, of which the most frequent is the contamination of drinking-water by leaden pipes or cisterns. Rarer causes are from snuff packed in lead paper and from certain hair dyes containing lead. A gouty tendency renders a patient more liable to lead paralysis; so also does alcoholism (see Vol. II. p. 64).

Symptoms. — The paralysis is usually preceded by other symptoms of lead poisoning such as colic, constipation, joint pains, or cerebral affection (encephalopathia saturnina). A blue line along the gums should always be looked for. Anæmia is the rule, while chronic nephritis and arterio-sclerosis are not infrequent.

Both upper limbs are usually affected, the right hand being attacked before the left in right-handed patients, and *vice versâ*. The onset of weakness is generally subacute, taking several weeks to develop.

The extensors of the fingers and wrist are generally the only muscles affected, and the extensor communis digitorum, or even a part of that muscle, may suffer alone. When the extensor paralysis of wrist and fingers is marked, wrist-drop results, and the fingers remain loosely semi-flexed. In fully-developed cases the long extensors of the thumb are also paralysed, whilst the extensor ossis metacarpi pollicis remains unaffected. The flexor muscles of the wrist and fingers escape, together with the supinator longus and triceps. Sometimes the atrophy spreads to the intrinsic muscles of the hand.

The affected muscles rapidly waste and exhibit the electrical reactions of degeneration. Occasionally a swelling develops on the dorsum of the flexed wrist-joint, probably due to distension and thickening of the carpal synovial sacs.

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An upper-arm type of lead paralysis, affecting the deltoid, biceps, and brachialis anticus, is rare.

Throughout the disease there is usually neither anæsthesia, paræsthesia, nor pain.

The lower extremities are rarely affected, but, when attacked, the peronei and long extensors of the toes suffer, whilst the tibialis anticus, although supplied by the same nerve, usually escapes.

Prognosis.—In acute cases of the disease the prognosis, as a rule, is good, but relapses are common if the lead poisoning be continued, and in them the prognosis is less favourable than that of a first attack.

The **treatment** consists in removal of the source of poisoning and in the administration of iodide of potassium. The alimentary canal should be cleared of any lead it may contain by means of saline aperients, such as sulphate of magnesia, whilst the paralysis is to be treated by electricity and massage.

Arsenical Neuritis

If arsenic be administered medicinally in excessive doses and for too long a period, as has occurred in some cases of chorea, arsenical neuritis may result. The poison may also gain access by inhalation from arsenical wall-papers or pigments (see Vol. II. p. 62).

A remarkable outbreak of neuritis has been recently observed in England amongst patients who were in the habit of drinking certain varieties of beer, often in quite moderate amounts. On investigation the beer was found to be contaminated with a perceptible quantity of arsenious acid, sometimes together with selenious acid. It appears probable that arsenic rather than selenium was the cause of the neuritis. The chief source of the arsenic in the contaminated beer was from the brewing sugar (either glucose or invert sugar), prepared from starch by the action of commercial sulphuric acid, which latter, being obtained from pyrites ore, often contains a considerable quantity of arsenious acid.

The **symptoms** of poisoning from such arsenical beer are similar to those of alcoholic neuritis, viz.—weakness of the muscles of the limbs, with tenderness on pressure and absence of knee-jerks, to which are superadded various cutaneous affections, such as pigmentation, keratosis, herpes, erythemata, bullæ, and falling out of the hair and nails. Œdema of the eyelids was also occasionally observed, and sometimes ulceration of the gums. Many cases showed *erythromelalgia* or "red neuralgia": a condition of the

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extremities, chiefly the feet, in which the limb becomes red and exquisitely painful, the congestion often going on to a dusky cyanotic appearance if the limb be allowed to hang down (see p. 382). Hyperæsthesia of the soles of the feet is also commoner in arsenical beer neuritis than in ordinary alcoholic paralysis.

The fact that arsenic may be administered medicinally for prolonged periods in cases of chorea, epilepsy, and other diseases, in quantities considerably larger than those detected in arsenical beer (generally from one-third to one-eighth of a grain per gallon), suggests that the alcoholic vehicle in which the arsenic is consumed by beer-drinkers increases its toxic effects.

In pure arsenical neuritis unconnected with beer, both arms and legs are usually affected, most intensely at the periphery of the limbs. Pains and paræsthesia in the hands and feet are followed by weakness and wasting of the muscles. Sometimes there is marked ataxy of the limbs. The nerves and muscles are tender on pressure and the electrical reactions of degeneration are present. Hyperæsthesia to pain may exist with diminished sensibility to touch in the feet and hands. Trophic changes, pigmentation, and eruptions in the skin are relatively frequent. The knee-jerks are lost.

The **prognosis**, as a rule, is good, although recovery may be slow.

The **treatment** should consist in the local application of massage and electricity, and in the internal administration of iodide of potassium, and salicylate of soda.

Beri-beri

This is a tropical variety of multiple neuritis which has a special liability to attack the vagus and phrenic nerves in addition to those of the limbs. It also tends to produce œdema of the connective tissues and effusion into the serous cavities of the abdomen and thorax.

The disease clings to buildings and ships which harbour the specific virus, but it is not directly infective from one patient to another. An outbreak of the disease is favoured by deficient ventilation, over-crowding, and bad food.

Symptoms.—After a premonitory stage of languor and malaise, lasting days or weeks, the symptoms of multiple neuritis set in and are exactly similar to those of severe alcoholic neuritis. In addition there is œdema of the face and limbs, with breathlessness and

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palpitation and a feeling of distress behind the sternum and in the epigastrium. There may be hydropericardium, hydrothorax, and ascites, but the œdema is relatively slight in the genitals. The urine is scanty. As the œdema of the limbs subsides, the muscles of the limbs are found to be atrophied. Death may occur from respiratory paralysis or from heart failure, often due to hydropericardium (see Vol. I. p. 358).

Post-diphtheritic Neuritis

This is equally frequent after slight as after severe cases of diphtheria. It comes on usually from two to three weeks after convalescence has begun.

Symptoms.—The first symptom is usually paralysis of the palate, evidenced by its deficient movement or immobility during phonation, by the nasal quality of the voice and by the regurgitation of fluids through the nose during swallowing. The eyes are next affected, the ciliary muscles being paralysed, hence there is loss of the power of efficient accommodation and impairment of vision for near objects. The pupils, however, retain their reaction to light and to attempted accommodation. The external rectus may become weak in one or both eyes. The pharynx may be paralysed, so that fluids tend to enter the air passages during deglutition. Laryngeal palsies are uncommon. In severe cases the trunk muscles, intercostals and diaphragm, may become affected. The cardiac nerves are generally more or less affected, so that the heart is feeble and irregular, and fatal syncope may result.

In the majority of cases the knee-jerks are lost even where there is no paralysis of the limbs. Sometimes symptoms of multiple neuritis in the limbs are superadded, symmetrical and affecting the legs more than the arms. They may persist for several months even when the palate, eyes, and pharynx have completely recovered, which they usually do in the course of a few weeks.

Prognosis and treatment.—Cases of post-diphtheritic palsy may be fatal from syncope, from paralysis of the respiratory muscles, or from exhaustion owing to inability to swallow enough food. If food gets into the air passages fatal pneumonia may result. All these dangers must be guarded against. Absolute rest in bed should be insisted on in every case. Nourishing food must be administered frequently and carefully, with the addition of alcohol if necessary. When deglutition is much affected it may be necessary to maintain the strength by nasal or rectal feeding.

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Strychnia should be administered internally, and if paralysis of the limbs be present this must be treated on the same principles as in other forms of multiple neuritis.

Septicæmic and Puerperal Neuritis

Multiple neuritis may follow any septic wound or injury, not necessarily a severe one. It may also follow septic cases of child-birth or abortion, or even an acute attack of gonorrhœa in either sex. The symptoms may exactly resemble those of alcoholic neuritis, or they may be more localised, affecting especially the median and ulnar nerves.

Malarial Neuritis

This is sometimes a sequel of malarial fever, but it may also develop without previous fever in patients who have been exposed to the influence of malaria.

Symptoms. — The weakness and wasting affect the legs, especially attacking the anterior tibial group of muscles. The knee-jerks are lost and the electrical reactions of degeneration are present. The upper extremities usually escape.

Treatment.—Locally the disease should be treated like any other form of multiple neuritis, and quinine and arsenic should be administered internally. The patient should be removed, if possible, from the malarial district.

Tuberculous Neuritis

The symptoms of multiple neuritis occasionally develop in the course of moderately advanced pulmonary phthisis. The lower limbs usually suffer alone. In rarer cases the upper extremities may be affected, sometimes on one side only, and the affected hand may show "glossy skin," and other trophic changes in the cutaneous structure.

Cachectic and Senile Polyneuritis

The vitality of the nerves being lowest at the periphery, general malnutrition may lead to multiple neuritis. Some of these senile cases have atheromatous changes in the arterioles which supply the nerve sheaths. Such cases exhibit an asymmetrical and irregularly distributed atrophic paralysis of the limbs. This may be associated with senile gangrene.

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TUMOURS OF NERVES

Tumours of nerves are divided into two classes:—"true" and "false," true neuromata being those in which new nerve cells or nerve fibres actually form part of the tumour growth, false neuromata being those in which the tumour essentially consists of non-nervous structures. *Ganglionic or true neuromata* are exceedingly rare. They occur chiefly in connection with the gangliated cords and plexuses of the sympathetic. Microscopically they contain ganglionic nerve cells, in addition to nerve fibres.

The vast majority of neuromata are of the connective-tissue type, occurring in the perineurium or epineurium. Such neuromata therefore are merely neuro-fibromata, neuro-sarcomata, or neuro-myxomata.

Nerve trunks may also become secondarily invaded by carcinoma, generally by a direct infiltration of the primary growth. Syphilitic growths are rare, except on the cranial nerves within the skull.

Neuro-fibromata may be solitary or multiple. They may occur in the course of any peripheral nerve, varying in size from a pin-head to that of an orange, or even larger. Cases have also been recorded in which such fibromata occurred not only on the peripheral nerves of the trunk and limbs but also in the walls of the stomach and intestine, in the mesentery and on the mesenteric branches of the sympathetic.

Tubercula dolorosa are minute neuro-fibromata affecting the subcutaneous branches of sensory nerves. They vary in size from a millet-seed to a pea. They are sometimes extraordinarily numerous, many hundreds occurring in the same patient. They are palpable through the skin, and generally tender to the touch.

Recklinghausen's disease is a term applied to a combination of multiple lesions occurring in nerve trunks and in the skin. Within the category of its cardinal signs are included multiple subcutaneous tumours on the nerve trunks, either neuro-fibromata or plexiform neuromata; tumours of the skin of the molluscum fibrosum type; and cutaneous pigmentation, either in small freckly spots or in larger patches. The bones are sometimes found, post-mortem, to be abnormally soft.

The disease, though probably congenital in origin, sometimes becomes rapidly progressive. The tumours on the nerve trunks increase in size and may produce fatal compression symptoms.

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Molluscum fibrosum is a term applied to multiple fibromata, sessile or pedunculated, affecting cutaneous nerves and larger nerve trunks, often congenital and associated with pigmented nævi. If a large area of skin be affected, it may form large pendulous folds. The commonest situation of a single pedunculated molluscum tumour is on the labium majus.

Plexiform neuroma is a twisted and tangled mass of interlacing cords on the course of a nerve, congenital in origin and found most frequently on some branch of the trigeminal nerve, although any nerve, cranial or spinal, may be affected. Its structure is that of a fibro-myxoma, traversed by the nerve fibres. It causes no symptoms beyond mechanical ones.

Symptoms. — From the above it will be understood that symptoms are often entirely absent in cases of neuromata, unless mechanical compression by the growth produces neuralgic or neuritic symptoms. In the latter case, paræsthesia and some blunting of sensibility in the area of the affected nerve may occur. Actual motor paralysis or muscular atrophy is rare. Sometimes a nodule growing on a spinal nerve root may compress the spinal cord and produce paraplegia.

Treatment is of little avail. If the tumours cause no symptoms they should be left alone. If a solitary tumour produces a pressure neuritis, it may be excised, and this can sometimes be accomplished by shelling the tumour out of the nerve sheath without dividing the nerve. Multiple tumours are best left alone.

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NEURALGIA

Paroxysmal pain in the area of cutaneous distribution of a sensory nerve, or along the course of a nerve trunk, has been termed "neuralgia." This may be symptomatic of actual structural disease of the nerves, or it may be that no such disease is discoverable. It is unfortunate that the laxity of medical nomenclature has included cases of both categories under the single term of "neuralgia." Strictly speaking, it is better to restrict the name as far as possible to those cases in which no recognisable microscopic changes are demonstrable in the affected nerves or nerve roots, and clinically to cases where structural disease can be reasonably excluded.

Etiology. — Neuralgia rarely occurs in childhood. It is commonest in adult life, and especially in middle-aged people. Males are affected more than females, though the liability in the female sex is increased during pregnancy and the puerperium, also at the climacteric. A neuropathic, gouty, or rheumatic diathesis can sometimes be traced. Neuralgia is most common in patients who are debilitated from any cause. After exhausting diseases, especially influenza and various fevers, and in conditions of anæmia, a patient is certainly more liable to neuralgia than when in good health. In a considerable number of cases the exciting cause is exposure to cold and damp, affecting especially the part where the neuralgia is afterwards felt. In other cases a source of peripheral irritation is present, as for example when a carious tooth or a refractive error in the eye causes trigeminal neuralgia, or when disease of an internal organ causes neuralgia of a limited cutaneous area. Toxic influences, such as alcoholism, lead poisoning, gout, diabetes, and malaria may induce neuralgia, but it should be remembered that such pain may sometimes be symptomatic of an early neuritis. Again, the neuralgia frequently present in cases of herpes is probably symptomatic of an organic affection of the sensory cells in the Gasserian ganglion or of the posterior nerve roots.

Symptoms. — In neuralgia the essential symptom is pain, which comes on in paroxysms lasting for periods from a second or two up to many minutes. The patient may describe the pain as "boring," "burning," "tearing," "shooting," etc. It may

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be superficial or deep. It often varies in intensity, even during the paroxysm. The most severe pain is that which occurs in trigeminal neuralgia. In the intervals between the attacks, the pain may completely disappear, or a constant dull aching may persist in the affected area. The frequency of the attacks varies in individual cases from one or two per diem to several hundreds. They may occur spontaneously, or may be excited by local stimuli, such as chewing, coughing, or sneezing. The pain is most intense along the affected nerve trunk or its branches, and at the height of the paroxysm it may radiate into adjacent nerve areas. It is rarely bilateral.

Reflex motor spasms often accompany the pain, as in trigeminal neuralgia, with its attendant facial spasm. Secretory phenomena, such as lacrymation and salivation, sometimes occur, and the skin may become locally pale or flushed. In long-standing trigeminal cases the hair may become gray or fall out, and the skin may be indurated. The affected area of skin is generally hyperalgesic, even between the paroxysms, and slight dulling of cutaneous sensibility may coexist ("anæsthesia dolorosa"). This latter, however, is always suggestive of a structural lesion.

Special "tender points" are to be found in most cases, situated over the points of exit of the nerves through bony foramina, or where the nerves cross rigid structures, such as bones or fasciæ, or over the vertebral spines corresponding to the origin of the affected nerves. In long-standing cases the patient's general nutrition is impaired, and he may become melancholic or even suicidal.

Diagnosis.—In a typical case this is easy, the paroxysmal pain confined to a particular nerve area, together with the "tender points," being quite characteristic. But in every case, before arriving at a diagnosis of idiopathic neuralgia, organic disease should be carefully excluded, since conditions such as tabes, aneurism, spinal or cerebral tumour may cause symptomatic neuralgia. The "psychalgia" of hysterical patients may somewhat resemble neuralgia. Such cases, however, are usually associated with other hysterical "stigmata," and, moreover, in them the pain tends to be more widely diffused, and may affect the entire half of the body.

Prognosis.—True neuralgia is an obstinate affection, lasting, as a rule, for months or years. The prognosis is somewhat better in young and well-nourished patients.

As to **treatment**, all sources of peripheral irritation should first be searched for, and, if possible, removed. The general nutrition should be improved by diet and tonics, and, if necessary, by massage.

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Stimulants should be most carefully regulated. In recent cases, especially with a rheumatic or gouty diathesis, diaphoretic treatment by hot baths, etc., yields good results.

During the paroxysms it may be necessary to administer morphia hypodermically, but the patient should not be permitted to do this himself, lest the morphia habit be acquired. Sometimes the local application of the constant current relieves the attack, the positive pole of the battery being placed over the painful area.

As a last resort, surgical interference may be necessary. Nerve stretching and nerve excision, as a rule, give but temporary relief for a few weeks or months. The most lasting results are obtained by excision of the corresponding sensory ganglion, either the posterior nerve-root ganglion, or the Gasserian ganglion, as the case may be.

Special Varieties of Neuralgia

Trigeminal Neuralgia—"Tic Douloureux"

Of all forms of neuralgia, that of the fifth cranial nerve is the most severe. It is also numerically one of the most common situations of neuralgia. It rarely affects all three divisions of the nerve; a single division or two adjacent divisions usually suffer. The supra-orbital division is more frequently affected than the two lower divisions. It is almost never bilateral, except in cases of diabetes.

Causes.—Local exciting causes are sometimes to be found in the mouth, nose, or eye, as for example carious or abnormally situated teeth, exostoses, nasal polypi, catarrh of the nose or of one of its accessory sinuses, refractive errors in the eye, conjunctivitis, iritis, etc. Sometimes neuralgia occurs in edentulous subjects. Such neuralgia has been ascribed to changes occurring in the alveoli. Many cases, however, depend on secondary alterations in the temporo-maxillary joint, and may be relieved by a well-fitting set of artificial teeth.

Symptoms.—During the attack there is intolerable agonising pain in the area of one or more divisions of the fifth nerve. It is usually sudden in onset. The patient presses his hand to his cheek and avoids all movement of the face or jaw, although strong involuntary spasm of the facial muscles on the affected side may occur. During the height of the paroxysm the pain may radiate into other branches of the nerve, or even into other adjacent nerves,

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as for example into the neck and shoulder. There is usually lacrymation, sometimes also excessive secretion of nasal mucus and of saliva, all on the affected side. In long-standing cases affecting the scalp, the hair may ultimately change colour, or fall out over the affected area.

The chief "tender points" are at the bony foramina through which the branches of the nerve emerge, at the supra-orbital foramen in the upper division, at the infra-orbital foramen in the middle division, and at the mental foramen, and where the auriculo-temporal crosses the zygoma in neuralgia of the third division.

CERVICO-OCCIPITAL NEURALGIA

In this variety, the pain affects the area supplied by the four upper cervical nerves, that is, the neck and the back of the head. The great occipital nerve (arising from the second cervical nerve) is more frequently attacked than any other nerve in this region. The "tender point," where the great occipital pierces the deep fascia, is midway between the mastoid process and the spine.

Cervico-occipital neuralgia is peculiar in being commonly bilateral. In some cases the carrying of heavy weights on the head is said to be an exciting cause. In every case, disease of the cervical vertebræ should be carefully excluded.

BRACHIAL NEURALGIA

As a rule this affects the whole area supplied by the plexus, comprising the four lower cervical roots, together with the first dorsal root. Individual nerves, such as the median or ulnar, are rarely affected alone. The pain affects the lower part of the neck, the shoulder, and the entire upper limb. It is usually made worse by movement of the limb, which feels heavy and numb, although there is no actual paralysis. If anæsthesia or trophic changes be present it is probable that some structural lesion, such as neuritis, exists. The "tender points" are over the musculo-spital, median, and ulnar nerves.

INTERCOSTAL NEURALGIA

In this variety the pain is distributed along the anterior branches of the spinal nerves. It is seldom confined to a single nerve, but affects several adjacent nerve areas. As a rule the pain is more or

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less continuous, with paroxysmal exacerbations. The corresponding skin area is hyperæsthetic, so that even the pressure of the patient's clothes may be painful. Anæsthesia is rare, and renders a diagnosis of structural nerve disease more probable than one of mere neuralgia. The "tender points" are diagnostically important. They are situated at the points of emergence of the three branches of the intercostal nerve, one near the spine, another in the mid-axillary line, and a third near the middle line in front.

Intercostal neuralgia may precede or follow herpes zoster, and may persist long after the eruption has passed away. The presence of herpes indicates that the pain is more than a mere neuralgia, and signifies some organic affection, probably of the spinal ganglion.

Symptomatic intercostal neuralgia may occur in various other organic affections of the spinal nerves, as, for example, in compression by an aortic aneurism, or in affections of the spinal meninges.

In diseases of the thoracic and abdominal viscera it is not uncommon to have cutaneous pain in small areas on the chest, abdomen, or limbs. The locality of the pain in such cases corresponds to the segmental spinal innervation of the diseased organ. Each viscus has sympathetic fibres belonging to a certain segment of the cord, hence visceral disease may cause reflex pain and hyperæsthesia in the sensory area of the corresponding segment of the cord.

MAMMARY NEURALGIA

This affects women, generally about middle life. It is often associated with anæmia or hysteria. There is great hyperæsthesia of the skin of the breast, and especially of the nipple, together with paroxysmal pain in the breast. Sometimes a milky fluid is secreted during or after an attack. The pain is usually worse during the menstrual period. The "tender points" are not in the gland itself, but over the corresponding dorsal vertebræ, from the second to the sixth.

LUMBO-ABDOMINAL NEURALGIA

This affects the lower half of the trunk. It spreads from the lumbar region forwards to the front of the abdomen, sometimes to the groin and genitals and downwards to the upper gluteal region. Occasionally during the paroxysm there is cramp-like spasm of the abdominal muscles and of the cremaster. Vomiting may also occur. The "tender points" are over the vertebræ, the middle of the iliac crest, the linea alba, inguinal canal, and scrotum or labium.

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NEURALGIA PARÆSTHETICA

This is a neuralgia of the external cutaneous nerve, affecting the outer aspect of the thigh. It is by no means rare. There may be paræsthesia and sometimes also a slight blunting of sensibility of the affected area in the intervals between the attacks. The pain is often induced by standing or walking, possibly from a stretching of the fascia lata. The "tender point" is at the anterior superior iliac spine.

ANTERIOR CRURAL NEURALGIA is rare, and OBTURATOR NEURALGIA still more uncommon. They may occasionally be symptomatic of a lumbar or pelvic tumour, and obturator neuralgia may be symptomatic of an obturator hernia stretching the nerve.

SCIATICA

The region of the sciatic nerve is by far the most frequent site of neuralgia. The term "sciatica," however, has unfortunately been made to include cases both of neuralgia and of sciatic neuritis. Moreover, all grades of so-called sciatica occur intermediate between neuritis and mere neuralgia.

Etiology.—Sciatica never occurs in children. It is a disease usually of middle life and affects men more frequently than women. A gouty diathesis is common; less frequently diabetes is present. Some cases of sciatic neuritis seem to follow myalgia in the lumbar region. The most common exciting cause is exposure to cold and wet, as by sitting on damp grass. Contusions of the sciatic nerve and compression by intra-pelvic growths, by scybalous masses, or by the pregnant uterus may also induce a secondary neuritis.

Symptoms.—There is usually a dull aching sensation in the back of the thigh, more or less constant, with occasional paroxysms of darting or boring pain, generally from above downwards, along the course of the sciatic nerve. Any movement of the limb which renders the nerve tense, or any local pressure, such as is produced by sitting on the edge of a hard chair, also brings on a paroxysm. The patient therefore keeps his hip and knee habitually slightly flexed on the affected side so as to relax the nerve. A slight degree of scoliosis is not uncommon, the concavity of the curve being towards the sound side. In some cases the pain is increased by walking; in others it is diminished. When the patient sits down, he rests his weight on the ischial tuberosity of the sound side, so as to avoid pressure on the sciatic nerve. The nerve is

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rarely tender in its entire course. The cremasteric reflex is sometimes exaggerated on the affected side.

The "tender points" are at the posterior iliac spine, at the sciatic notch, at the back of the knee, below the head of the fibula, and behind the malleoli. Any movement which stretches the nerve increases the pain. Thus an excellent test for sciatica is to flex the hip passively with the knee extended. Blunting of sensibility in the areas of distribution of the posterior tibial or peroneal nerves is rare. Sometimes in old-standing cases there is a degree of atrophy of the muscles supplied by the nerve.

Any alteration in the electrical reactions of the muscles indicates an actual neuritis; so also does the existence of anæsthesia. In cases of sciatic neuralgia the knee-jerk on the affected side is usually brisk, whilst in sciatic neuritis it may be diminished or absent.

TESTICULAR NEURALGIA

Pain in the testis and spermatic cord, with cutaneous hyperæsthesia, is associated with so much mental depression that it merits special mention. During a paroxysm there may be contraction of the cremaster on the affected side, and sometimes vomiting.

A certain number of cases are associated with varicocele, and if the latter be treated by a suspensory bandage, or by surgical methods, the condition may be cured.

COCCYGODYNIA

Neuralgic pain in the region of the coccyx is almost exclusively confined to the female sex. The pain may be spontaneous, or it may be induced by sitting or walking, or when any of the muscles attached to the coccyx are brought into action, as in defæcation, or sometimes even on micturition. The coccyx is tender on pressure. Local disease of the coccyx or of the adjacent intra-pelvic structures should always be carefully eliminated. The actual paroxysm is generally relieved by a morphia suppository.

PURVES STEWART.

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FUNCTIONAL DISEASES OF THE NERVOUS SYSTEM

While the rapid strides which science has made in the direction of more perfect methods of investigating the finer structure of the elements of the nervous system have thrown much light on the nature of many diseases, there still remain several maladies in which a primary structural change in any of the nerve elements is as yet undiscovered. Such are conveniently, if somewhat arbitrarily, classed under the term FUNCTIONAL DISEASES. The hitherto negative results of pathological investigation in these conditions have many explanations, for finer structural changes may be too delicate for observation by the methods at present at our disposal; and, further, so incomplete is our knowledge of the anatomy, and more especially of the physiological functions and mutual interactions of the higher elements of the nervous system, that it is sometimes difficult even to presume with any degree of correctness what elements are at fault. Since the function of living tissue is the expression of its metabolism, all deviation of function from the normal must be accompanied by alterations in the chemical process of the elements, and it is obvious that when disorder of function is primarily dependent upon such chemical changes, the latter, from their high intricacy, are likely to permanently elude the search both of the chemist and the pathologist.

The inclusion of a malady under the group of functional diseases is somewhat arbitrary, and it is probable that in the course of scientific progress some of the diseases so included will be relegated to the position of organic diseases of known pathology. Indeed, in the case of chorea, which is here included, this event is in progress. On the other hand, it is not likely, from the nature of the disease, that a primary structural pathology will ever be established in such diseases as epilepsy and migraine. When considered broadly in terms of disordered metabolism of nervous elements, functional disease implies perversion rather than abrogation of function, and the conception entails the possibility on the one hand of the rapid appearance and equally rapid disappearance of the functional disorder; on the other hand, of life-long perversion of function, for action in any direction induces a change in molecular constitution which predisposes to and facilitates the

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repetition of such action. Habit becomes impressed as indelibly upon the individual nerve elements as upon the man, and upon this fact depends the high perfection of function that may be attained by frequent repetition. Functional disease concerns primarily the higher parts of the nervous system, presumably in many diseases the highest part of the nervous system, and as according to the doctrine of Dr. Hughlings Jackson the lower nervous system (spinal cord and medulla), and therefore the body, is represented by each part of the higher nervous system from basal ganglia to frontal lobes in increasing compound proportion, the term implies the possibility of the universal distribution of symptoms. Thus the complex tangle of symptoms which may occur in hysteria becomes more conceivable; in this disease there is hardly a function of the whole body which may not be deranged.

While the absence of primary structural change has been mentioned as the boundary of so-called "functional disease," it must be pointed out that the latter may involve obvious organic change in the tissues, and, further, that the early symptoms of structural disease may be indistinguishable from those in which no structural change has hitherto been discovered. These somewhat conflicting statements require further explanation. The disorder of function of a nerve cell is accompanied by a deviation of its intracellular chemical processes from the normal, and such abnormal metabolism is likely to lower the tenure of vitality of the cell, and consequently the cell may undergo premature decay. Such secondary structural degeneration is not uncommonly found in functional disease; in epilepsy, for example, degeneration of the cerebral cortex is found in proportion as the disease is of long standing and the attacks frequent and severe. Again, if we imagine a slow structural change involving the nerve elements, for instance, alterations of blood supply or pressure from sclerosis, such a change must involve alterations in the metabolism of the elements, and disordered function may arise, comparable to, indeed exactly similar to, that produced by diseases which we call "functional," before any signs of organic disease are apparent; for instance, in the early stages of disseminated sclerosis a monoplegia or paraplegia with anæsthesia may appear, rapidly sometimes, as the result of mental shock, to disappear equally rapidly, in every way resembling a "functional" phenomenon. In this disease the appearance and disappearance of symptoms may occur several times, but though the symptoms of the first manifestation may disappear completely, those of the second leave, as a rule, some slight permanent disability—the

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organic remnant which is added to by each fresh occurrence of symptoms, until the "functional" aspect of the malady is changed to one of structural disease. So intimately are the various elements of the higher nervous system interdependent in their physiological action, that disease of one part of the nervous system may produce widely-spread functional derangement in elements not directly involved. As an instance, the most typical symptoms of hysteria may occur in association with cerebral tumour.

It has been above asserted that all functional disease is dependent upon disordered metabolism in the nerve elements. Are not all structural diseases of the nerve elements similarly dependent upon primary metabolic derangement? This question we must answer in the affirmative, and at this point functional and organic disease run into the same line. There is no fundamental pathological distinction to be drawn between them. To argue an essential difference in the primary metabolic disorder in the two cases merely begs the question. In the diseases which we term organic the gross structural change follows the nutritional change sooner and in greater degree; in functional diseases where symptoms are persistent, later and in less degree. In the latter the possibility of a return to normal function is great in inverse proportion to the duration of symptoms, or as it depends upon the individual peculiarities of the disorder of function.

The conditions first described in the following section, headache and vertigo, though in reality symptoms of disease, are often so much more obtrusive than their cause as to deserve separate description. Vertigo exemplifies the widely-spread disturbance of the function of equilibration which may be produced by change in the physiological action of any part of the complex nervous apparatus subserving that function, for it occurs in derangement of the oculo-motor mechanism, of the membranous labyrinth and of the cortex cerebri, and in the latter connection is frequently associated with epilepsy as a premonitory symptom. The intimate functional association of the various parts of the nervous system (in this case that of the equilibratory and splanchnic systems) is suggested by the common occurrence of vomiting in vertiginous attacks. The constant presence of some degree of impairment of consciousness in the attacks is important as showing the profound effect upon the higher nervous system of disturbance of one group of its elements. Headache, a symptom of disease or derangement of so many systems of the body, deserves consideration among functional diseases of the nervous system, for we are entirely ignorant of the

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nature of the processes underlying its production. Migraine is perhaps the most striking example of a "functional" disease. The influence of heredity upon its incidence, its frequent persistence from early childhood to old age, with preservation throughout life of the highest standard of cerebral function, and the absence of known structural changes in this disease, suggest a transmissible peculiarity in certain elements of the nervous system which, while allowing of temporarily disordered liberation of energy in these elements, neither entails functional incapacity, nor do the temporary disturbances ever, when often repeated, involve functional or structural deterioration. Epilepsy, to which the last-mentioned disease is in some of its features allied, evidences the self-perpetuating character of disordered function in unstable nerve elements, for with each exhibition of spontaneous liberation of energy the resistance or self-control of the nervous elements is lessened and their capacity for discharge increased. In the majority of functional diseases of the nervous system the condition of instability of the nervous elements is probably a most important factor, and the disorder of function tends to work in a "vicious circle," as, for instance, in the diseases habit spasm, hysteria, and torticollis. In epilepsy, however, the disturbance of function, at first paroxysmal, usually involves an increasing deterioration in the functional capacity of the higher nervous elements, and this is more marked if the incidence of the disease is at that period when the nervous system is in the most active period of its development, and is greater when the paroxysmal disturbance is more frequent. In long-standing cases of this disease structural changes in the cerebral cortex are often found, and for this occurrence two circumstances are probably responsible: first, the severe cerebral congestion which occurs in the asphyxial stage of the epileptic attack may occasion vascular lesions of the brain; and, secondly, the tenure of natural vitality of the affected elements may be shortened by the alteration of nutrition which the functional derangement involves. Cortical fits due to organic disease of the brain are, from the similarity of the phenomena, necessarily described with epilepsy. The convulsions of idiopathic epilepsy and those due to organic disease are sometimes identical, and it is only to be expected that this should be so, for the functional derangement, in the former case inherent in the nervous elements, may be produced in the latter case in the same elements by presence of a gross lesion in their vicinity.

Infantile convulsions and tetany are excellent examples of the effect of mal-nutrition or perverted general metabolism upon the

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nervous system during its most active period of growth. The stability is diminished, and spontaneous discharge or increased reflex excitability results.

The manifestations of hysteria, extending from conditions closely simulating those due to structural disease to such as are definitely conditions of mental derangement, are by some authorities termed "functional" disease to the exclusion of other maladies. This arbitrary use of the term, however, for a clinical group deprives it entirely of its etymological meaning, though it avoids the stigma which is attached by the laity to "hysteria." The relationship of this disease to organic nervous disease on the one hand, and to insanity on the other, is shown in its neuropathic heredity. That form of the disease in which hysteroid attacks occur seems to be closely allied to epilepsy, and Sir William Gowers has recorded cases in which diagnosis between these two conditions was impossible. Though the variety of symptoms of hysteria is very great and the simulation by it of other diseases very considerable, yet certain features are fairly constant, and their absence should exclude the diagnosis. Hysteria is not a "pigeon-hole" in which to thrust all ill-understood nervous diseases. The rapid appearance and sudden disappearance of symptoms reveals in a more striking manner the functional nature of the malady in some cases of hysteria than in any other disease here classed as "functional."

The influence of disorder of nerve function upon general metabolism in neurasthenia is extremely marked, and it has been theorised that an auto-intoxication as a result of such disorder occurs in this disease. The psychic state approaches hypochondriasis, self-consciousness is greatly increased, and subjective sensations form a great part of the symptomatology.

It has been above stated that the pathological anatomy of chorea is in process of being firmly established, yet movements occur in cases undoubtedly hysterical, which are indistinguishable from those of true chorea, and on the other hand hysterical manifestations may occur in rheumatic chorea. It would appear that there is sometimes a double factor in the production of chorea, an element of functional instability of the nervous elements and the rheumatic poison. The incidence of chorea in rheumatic subjects who have neuropathic heredity is far greater than when there is no such nervous taint.

The occupation neuroses stand in a special position among the functional diseases, in that they are disorders occurring from the prolonged exertion of a particular function. The break down of a

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special group of nerve elements produces paralysis in effect for a certain complex act alone. Such neuroses strikingly illustrate the persistence of functional perversion, and suggest strongly the involvement of the higher parts of the nervous systems. A patient with writer's cramp once well established is hardly ever cured. He learns to write with the other hand, but the functional disorder follows. He uses the typewriter to overcome his difficulty, but in this act too the functional disturbance follows and typist's cramp comes on. It is here the cortical centres for expression in writing which are at fault, and these centres come into action whether the right or left hand be used, and either for the pen or for the machine. There does not seem to be in these disorders primary instability of the nervous elements, as is the rule in the other functional disorders; the defect is produced by prolonged over-exertion, and neuropathic heredity, so commonly present in the latter, is rare in the history of occupation neuroses.

Paralysis agitans, here included from the absence of structural changes, illustrates forcibly the limitations of our present methods of examining the finer structure of the nervous system. A chronic degenerative condition of insidious outset and progressive course, leading, in the course of time, to complete incapacitation and showing no tendency to even temporary improvement, yet gives no evidence of structural change. It is, however, probable that such will be found with improvement in the method of pathological research. Clinically, too, it stands apart from the other conditions described in this section.

It will be seen from the above that many of the functional diseases possess etiological relations and symptoms in common, and that they may be associated one with the other. Thus hysteria and epilepsy and insanity are closely associated; chorea is not infrequently associated with hysteria and with epilepsy, habit spasm with hysteria, and torticollis with hysteria and insanity. On the other hand, the association of neurasthenia and paralysis agitans with other of the functional diseases is extremely rare.

J. S. COLLIER.

HEADACHE

By this is understood pain of a more or less diffused character felt in some part of the cranial region. It is to be distinguished from a merely localised pain, although the two may co-exist, and

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also from neuralgic affections of the head, in which the pain is experienced along the course of particular nerves, such as tic douleureux, supraorbital neuralgia, etc.

It is one of the most frequently occurring symptoms, and is associated with diseases of the most varied character, from which circumstances it may be that its clinical significance, whether from a diagnostic or prognostic aspect, may be comparatively slight.

Before attempting an explanation of the pathology of this symptom, it may be well to indicate the several morbid conditions with which it is commonly associated and to that extent may be regarded as its causes.

(α) *Diseases of the brain and its membranes.*—Almost any affection included within this group is liable to be marked by headache more or less severe, the most frequent being tumour, abscess, and meningitis; less constantly hæmorrhage, embolism and thrombosis of the cerebral vessels and chronic degenerative changes.

Abnormal vascular conditions of the cranial contents, such as congestion, as from obstructive heart disease or pressure on the large venous trunks in the thorax, and increased arterial tension from general arterio-capillary fibrosis are usually characterised by headache of variable intensity and persistence. It would appear that many headaches depend upon a want of adjustment, or of balance in the cerebral circulation; there being a state of spasm in the vessels of one area and of dilatation in those of other regions.

(β) *Toxic states.*—The introduction into the body of certain poisonous substances of the most varied character, such as alcohol, lead, nitroglycerine, thyroid extract, etc., is very liable to give rise to headache among other effects; and a similar result follows those conditions of auto-intoxication which arise in the febrile state or from abnormal fermentations in the gastro-intestinal tract, such as occur in many forms of indigestion and constipation. In the same way is perhaps to be explained the headache of anæmia.

(γ) *Nervous headaches* are those referable to states of the nervous system, though no definite lesion has yet been discovered. Such are the headaches of exhaustion, of neurasthenia, and of hysteria.

The pain, often agonising and long-continued, of migraine, or of neuralgia of the scalp, is not strictly to be included within the range of headache, inasmuch as the area of distribution of the pain corresponds to that of a special nerve or nerves and is not of the diffuse character so essentially a feature of headache proper.

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(δ) *Peripheral irritation.*—The effect of this in producing headache is undoubted, and the site of irritation is most varied. Among the commonest are (i.) the teeth, caries of which, or their eruption, especially of the last molars, are frequent causes. (ii.) The eye, especially prolonged strain, as from sight-seeing, glare of sun or other strong lights, and errors of refraction, astigmatism or grave structural lesions causing increased ocular tension, such as glaucoma. (iii.) Diseases of the external auditory meatus or of the middle ear. (iv.) Affections of the nasal and connected cavities and throat, adenoids, tonsillitis, etc. (v.) Visceral diseases, of which perhaps ovarian are the most frequent.

(ε) Certain *atmospheric states*, especially thunderstorms, or a low barometer, appear to be directly responsible for headache in some persons.

Two expressions in common use, "a sick headache" and "a bilious headache," are extremely vague in their application. Strictly speaking the former may be said to be synonymous with migraine, which is characterised among other features by severe headache, which usually terminates in an attack of vomiting. Both terms, however, are commonly employed to denote headaches associated with sickness that are referable to indigestion, especially an acute gastric irritation from over-indulgence.

This arrangement does not claim to be either exhaustive or precise, but merely disposes in groups the chief morbid states with which headache is commonly associated. To make the arrangement complete, it would need to enumerate almost every known malady, for there is scarcely one that may not be accompanied by headache, in a more or less causal relation. So much depends on the individual temperament or general state of health, for whilst one person may hardly know from experience what headache means, in another it may be induced on the slightest provocation, and may even be almost a chronic condition. Nor is the grouping exact, owing to our ignorance as to the intimate pathology and immediate causation of headache. Thus headaches classed as toxic may some of them really depend upon vascular conditions of spasm or relaxation brought about by the poisons, rather than by their direct effect upon the cortical cells, and this is especially likely to be the case with the headaches which follow the administration of the nitrites.

The conscious appreciation of pain, as of other sensations, depends on the integrity of the cerebral cortex, "which contains, as is known, fields for the termination of, so to say, each avenue of

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sense" (see p. 25),[1] though how far special "pain centres" exist distinct from those of common sensation is an open question. Yet injury or electric excitation applied to the cortex itself does not produce pain; and, as Professor Sherrington points out, a "pain aura" hardly ever occurs in epilepsy, "although other orders of sensation—visual, auditory, tactual, thermal, gustatory, and odorous —seem each laid under contribution." To evoke a painful sensation in the cortical cells "excessive" stimulation of the nerves of "common sensation" is required (see p. 5), special pain nerve fibres and end organs not being apparently necessary. Although for the production of pain centripetal impulses are requisite, it may be that "bodily pain is elaborated in the Rolandic region and that thence are initiated emotional movements, respiratory, vascular, facial, etc., and it has been suggested that the severe pains of hysterical and hypochondriacal patients are in some instances of cortical origin." The ability which appears to be possessed by exceptional individuals of inhibiting the sensation of pain would accord with this.

Applying these considerations to headache, it is permissible to conceive of this symptom being due (*a*) to intrinsic nutritional disturbances in the cortical cells over a wider area, as in the headaches of neurasthenia and hysteria, though the excitant of such disturbances is not yet apparent. The circulation to the cells of a blood containing effete or poisonous material may be regarded as a cause of perverted metabolism, and so far furnishes an explanation of toxic headaches. (*b*) Mechanical irritation of the cortical cells by the pressure of over-full cerebral blood-vessels. (*c*) Peripheral irritation of various afferent nerves of common sensation, whether from the meninges, or the blood vessels (nervi vasorum) of the brain, from the scalp, especially the muscular aponeurosis, or from extra-cranial sources, as eyes, teeth, viscera, etc., so stimulating the cortex cells as to produce headache. But the question at once arises, assuming that their several regions—meninges, vessels, scalp, special sense organs and viscera—are "represented" in the cerebral cortex, to which of the cranial structures is the pain of headache to be referred, what, in short, is the aching structure? To this no satisfactory answer can be returned. Yet it would seem to be in accordance with what is known of pain elsewhere that the pain sensation excited in the cortical cells by centripetal stimuli of sufficient intensity should be referred to some region. For when

[1] See also "Cutaneous Sensations" by Prof. Sherrington, F.R.S., in Schäfer's *Text-Book of Physiology*, vol. ii. p. 20 and *seq.*, from which the above quotations are made.

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any part of the body is spoken of as the seat of pain, what is meant is that the irritant in that part so stimulates the sensorium (cortex) as to cause pain which is at the same time referred to the seat of irritation. In headache, is the seat of irritation the scalp and muscular aponeurotic structures, the nervi vasorum of the brain or the meninges, and is it to either of these that the characteristically diffuse pain of headache is referred? And, if so, which is involved in the headache caused by irritation of peripheral sense organs (eye, ear, nose, etc.) or viscera?

Some importance as a means of ascertaining the cause has been attached to recognition of the *situation of the headache*, and very often it may be helpful, though not invariably reliable. Probably the greater number of headaches are *frontal*, certainly those of toxic origin are usually so, and this is true of most dyspeptic headaches, as it is also when the symptom is connected with malaria. Affections of the nasal cavities and communicating sinuses are likely to cause headache in this situation, as Sir Lauder Brunton says, at the margin of the hairy scalp. When attributable to neurasthenia, to anæmia and fatigue, to eye strain or to congestion of the brain and cranial structures, the headache is often frontal, though not infrequently general in distribution. The commonest cause of *occipital* headache is constipation, and may then be associated with pain in the frontal region. Congestive headaches are also frequent in this locality. Caries of the lower molar teeth is said to induce occipital headache on the same side. Similarly, when the ache can be referred to the vertex, the incisors have been found decayed (Brunton). The headache of anæmia, and debility, and of the climacteric is often felt most severely in this region, as also is the "clavus" of hysteria. Headaches limited to the temporal region are usually neuralgic or migraine; eruption of the lower wisdom teeth or carious molars may lead to the same, and when bilateral the cause is probably toxic.

Too much reliance must not be placed on these distinctions nor on the seat of the symptom as an indication of the situation of a cerebral tumour, though this may be so, especially if there be tenderness on deep pressure over the area (see p. 49).

The *character of the pain* is most variable and is only imperfectly described by such conventional terms as dull, aching, throbbing, cutting, etc. It may be continuous with or without exacerbations, or intermittent. Posture may increase or diminish it, and this is more particularly the case with congestive headaches, which are usually worse on stooping or when the head is low. It is impossible

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to give any measure of the severity of the different forms of headache; it varies much with the cause, with the individual, and even in the same individual at different times. It is generally considered that the pain of cerebral tumour is the most intense of this class, and is said to be peculiar in preventing the patient sleeping, a state that frequently supervenes in other, even very severe, headaches. The diagnostic value of the symptom is much enhanced by its association with such conditions as pyrexia, delirium, tenderness of the scalp, photophobia, vomiting, etc.

The **treatment** of headache resolves itself into the removal of the cause as far as possible and special measures for the relief of the symptom. Of the former detailed description here is not required, and remedial agents may be conveniently grouped as anodynes and sedatives, those modifying the state of the vessels, and antidotal drugs. It is very uncertain, however, how far these several remedies act solely as grouped, and whether the same drug may not produce its beneficial effect by simultaneously controlling the vascular supply and allaying the irritability of the nervous structures, or neutralising the toxic effect of some poison. It would be impossible even to enumerate all the various drugs that have been recommended; only the general principles regulating the administrations of these several kinds of remedies can be referred to.

Of sedatives none are so generally useful and permissible as the bromides; together with rest, which the patient will naturally take, these drugs are probably the most reliable at our disposal. Their efficacy probably depends on diminishing the excitability of the cortical cells, perhaps also by regulating the cerebral circulation. Headaches that are classed as nervous are specially benefited by full doses of the bromides, which also afford considerable relief in the headache accompanying the onset of the acute infections. Phenacetin, antipyrin, and similar drugs are very variable in their effect on different individuals, acting almost like a charm with some and being almost useless with others.

When the condition is due to anæmia, neurasthenia, or fatigue, tea or coffee, caffein, guarana, or alcohol will give temporary relief, but should not be employed at the expense of the remedies appropriate to the causal disease.

It is seldom that opium or morphia are required, drugs that are of themselves not unlikely to cause headache; their careful use, however, in certain intracranial states, when the pain is agonising, may be permitted. Cannabis indica has been successfully used in migraine, as also croton chloral.

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Local anodynes are often very beneficial, and of these both heat and cold as applied by hot fomentations, hop pillow, sponging the face with hot water, ice-bags, volatile sprays, evaporating lotions, pigments composed of menthol, chloral, and camphor, are those most frequently employed, and may be supplemented by applications of belladonna or aconite.

The cranial circulation is also capable of regulation by hot or cold applications, by local bleeding, as leeches to the temple, hot foot baths, sinapisms, and blisters to the nape of the neck. The headache of a high tension pulse is usually relieved by nitroglycerine or other nitrites, and very often by the dilute nitric acid which is a valuable remedy in such cases. Small doses of iodide of potassium with or without bromides, and calomel followed by saline aperients effect the same result. Such drugs as strychnine, digitalis, caffein, and alcohol, by raising the arterial tension, may be indicated for the headache due to that state of the circulation.

A calomel purge and saline aperient is also the best remedy for most headaches due to errors of digestion. Five grains of antipyrin in an effervescing granular form is most useful for the same purpose. When the headache due to this cause is more chronic, and not simply the result of an excessive or indigestible meal, repeated small doses of calomel ($\frac{1}{4}$ to $\frac{1}{2}$ gr.) three times daily for several days may be tried, or the amount of meat food much diminished or entirely replaced by a vegetarian diet.

The headaches of syphilis are usually benefitted by iodide of potassium, as are those of malarial origin by quinine, and the "rheumatic" by salicylates, to each of which the bromides are useful adjuncts. The excruciating headache of cerebral tumour is found to be most amenable to the coal-tar products, of which antifebrin is the best, combined with iodide of potassium, 10 grains of the latter with 5 of the former; the necessity for morphia in such cases has been alluded to. Complete relief usually follows trephining the skull and so relieving the intracranial pressure.

W. H. ALLCHIN.

VERTIGO

SYN. GIDDINESS, DIZZINESS, SWIMMING IN THE HEAD

Disordered sensations of equilibrium arising from very varied causes are included within this term.

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The term includes several distinct forms of sensation, any or all of which may be present at the same time :—

(1) Apparent movement of objects, usually horizontally around the patient as a pivot, usually spoken of as the "room going round." Occasionally the apparent movement is around an axis on which he stands, the floor or pavement rocking like the deck of a ship.

(2) Sense of falling, either forwards, backwards, or to right or left. During the attack he staggers to recover his balance, or clings to some support, which often leads to unfounded charges of drunkenness.

Vertigo is only a symptom, and the cause must be looked for—

(*a*) In the peripheral organs concerned in the maintenance of equilibrium, especially the ear and the eye.

(*b*) In functional or organic affections of the central nervous system.

(*c*) In toxic conditions of the blood due to dyspepsia, gout, anæmia, etc. They probably produce vertigo through the central nervous system.

In practice the most frequent forms for which advice is sought are those due to dyspepsia, and to wax in the external meatus.

AUDITORY VERTIGO.—This form is usually associated with deafness. It may be due to the presence of insects, foreign bodies, or inspissated wax (most frequently the latter) in the external meatus; to middle-ear disease; or to disease of the labyrinth. Deafness is associated with the vertigo in nearly all the forms, but in the *labyrinthine* form bone conduction to the vibrations of a tuning fork is impaired or lost, while it is unaffected in vertigo due to disease in the middle or external ear. When the vertigo is due to disease of the labyrinth, the symptoms are usually complicated by subjective auditory sensations, such as hissing, buzzing, or ringing noises in the head (see Ménière's disease, p. 243).

Auditory vertigo is rarely constant, but comes on in paroxysms, which are often very severe, causing the patient to stagger, and even to fall suddenly. Mental disturbance is very apt to come on with labyrinthine vertigo.

Treatment.—Removal of wax, etc., from the meatus will cure many of the cases. Cases of labyrinthine vertigo are very intractable. A combination of the iodide and bromide of potassium gives relief more frequently than other drugs, and next in efficiency come quinine, jaborandi, and hydrobromic acid.

OCULAR VERTIGO.—Vertigo is occasionally caused by eye-strain,

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especially in myopic subjects. It may also occur when there is diplopia due to strabismus. Correction of the error of refraction with glasses, in the first case, or covering the affected eye with a shade in the other, will remove the troublesome sensation.

VERTIGO OF CENTRAL ORIGIN.—Vertigo is extremely common at the time of onset of an attack of hemiplegia, and when this is due to thrombosis there is often vertigo for several days previously. It is also a frequent symptom of intracranial tumour. It has little localising value, but is probably most common with tumours of the frontal lobe, and of the cerebellum. It is also a very common complaint of neurasthenic patients.

GOUTY VERTIGO.—Occurs usually in association with general arterio-sclerosis. It is probably sometimes due to the vascular condition, sometimes to the irritation of the central nervous system by uric acid or other substances in the blood. Most relief is afforded by careful dieting and the administration of salicylates followed by iodide of potassium.

GASTRIC VERTIGO.—Vertigo is very frequently the result of dyspepsia, or of indiscretions of diet. It is uncertain whether it is brought about by reflex action or whether it is caused by the formation of toxic substances in the alimentary canal which are absorbed into the blood, and act on the central nervous system.

Vertigo may also be caused by drugs. The susceptibility of individuals in this respect varies. Quinine, salicylates and salicine, cocculus indicus, and alcohol are the chief drugs which have this effect. It is also frequently met with in association with aortic disease.

MIGRAINE

SYN. HEMICRANIA, SICK HEADACHE

A paroxysmal nervous disturbance in which the prominent symptom is headache, usually attended with nausea and vomiting, and in some cases preceded by disorders of vision.

Etiology.—The disease usually makes its appearance in later childhood, and cases commencing after thirty are rare. It is rather more common among females than males, and is strongly hereditary, and may affect all the members of a family. It is common to meet with a family history of other neuroses, and often of gout and of trigeminal neuralgia. No exciting cause for the first attacks can

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usually be traced, but their subsequent frequency and severity is greatly increased by worry, overwork, etc., especially if the work be performed in bad hygienic surroundings. They are also aggravated by any disturbance of the general health, and especially by eye-strain, carious teeth, pharyngeal adenoids, menstrual disorders, or indigestion.

Symptoms.—The attacks most frequently begin in the early morning, and last from twelve to twenty-four hours, sometimes longer. Premonitory symptoms are not uncommon, and may be noticed over-night. For some hours previously the patient may feel "better than he has felt in his life," or he may have somnolence, slight headache or visual prodromata. In more than half the cases headache is the first symptom. This usually begins as a slight pain across the forehead, and is accompanied by mental irritability and depression. The pain soon becomes localised to one spot, usually just above one eyebrow, and is of a deep boring character. It increases and spreads until it extends over the whole of one side of the head (more frequently the left) and may pass down the neck to the arm. More rarely it spreads to the other side of the head. The nerves of the scalp are usually tender, on pressure, during the attack. The pain, though intense and causing much physical prostration, does not usually interfere with sleep. It may last any period from ten to twenty-four hours, or even longer. Nausea is usually present, increasing as the pain increases, and terminating after some hours by vomiting, after which the pain is usually much relieved and the attack ceases. The vomited matter often contains bile which has been regurgitated from the duodenum, and hence attacks of migraine are frequently spoken of as bilious attacks or more briefly as "liver." During the early stages of the attacks well-marked changes in the circulatory system occur. There is general arterial spasm, the face is pale, the hands and feet are cold, the pulse is slow, and the arterial tension is raised, especially in the temporal arteries. Shivering is common and tingling, numbness and other paræsthesia may be present on the affected side.

The visual phenomena which precede or accompany the appearance of pain are extremely interesting, and form one of the most characteristic features of the disease; they occur, however, in only one half of the cases. When present, they form the earliest symptom of the attack. The disturbance may consist in partial loss of sight or spectral appearances (teichopsia) or both. The unilateral character of the visual symptoms is always manifested as an affection of the corresponding halves of both fields of vision, and

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the loss of sight is always imperfect. There may be sudden general dimness of vision, or hemianopsia, or the change consists in a spot of dimness of sight, often with a bright central point which gradually increases in size and spreads towards the periphery. The spectral figures, when present, appear in the dim field; most commonly a bright spot appears, gradually expanding; its centre becomes dim, and the resulting ring, still expanding, loses its regularity and assumes a zig-zag shape with sharp, prominent, and re-entrant angles. This is known as the "castellated" or "fortification" spectrum. The ring then becomes dim at one part and disappears, the remaining part becoming lighter and often limited by colour. Meanwhile, within the ring light spots, in rapid movement or showers of stars, are commonly seen. The hemianopsia may persist for some time after the disappearance of the spectre. In other cases the visual phenomena are slight and are confined to a few sparks or flashes of light. In rare cases hallucinations of hearing and sight have been present, and sometimes vertigo, temporary aphasia, or even passing mental disturbance; in other cases motor symptoms in the limbs, usually confined to transient weakness or cramps, are present during the attack.

The symptoms are generally much intensified by light or noise, and the sufferer lies in a darkened room refusing to see any one.

The degree of severity of each attack varies from slight unilateral headache unassociated with other phenomena, and extending over an hour or two, to the most painful and prostrating disturbance, lasting from twelve to sixty hours.

The attacks tend to recur with more or less regular periodicity during the first half of life, but afterwards there is a tendency to spontaneous disappearance. They do not bring about any mental deterioration, and in fact are more common in persons of mental activity and high intellectual attainments.

Pathology.—No anatomical changes are known to underlie the phenomena of migraine, and the nature of the malady is a matter of inference only, while, from the nature of certain of the symptoms, its seat is in all probability the cerebral cortex. From the conspicuous pallor of the face and extremities, and from the raising of the arterial blood pressure during the attack, it has been suggested that spasm of certain cerebral arteries is the cause of the early symptoms, and subsequent vaso-dilatation in the same area the cause of the headache. According to this hypothesis, the disease is dependent upon derangement of the sympathetic nerves. There are many objections to this theory, for, in the first place, spasm of

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peripheral surface vessels does not argue a similar condition in the internal organs, and it is certain that the sympathetic nerves have very little effect upon the cerebral vessels and circulation; it is, moreover, not only unproved, but in the highest degree unlikely, that vaso-motor spasm can cause a deliberate, uniform, and peculiar train of phenomena, such as migraine. A more probable theory, put forward by Dr. E. Liveing, attributes migraine to a primary derangement of the nerve cells of the brain, the function of which is apt to be disturbed from time to time in a peculiar manner, the visible vaso-motor disturbance being a secondary phenomenon, the phenomena of migraine being thus analogous to those of epilepsy.

Some writers consider that an excess of uric acid in the body is an important exciting cause of the attacks, and the malady is not uncommon, at least as regards the peculiar headache, in chronic Bright's disease, and also in association with those vascular changes which are recognised as supervening upon prolonged mental anxiety and distress, especially in women.

Treatment—(*a*) *During the attack.*—If the bowels are confined a Seidlitz powder or some other quickly acting aperient should be taken. The patient should be kept in the recumbent position, and great relief is afforded by the application of hot bottles to the feet, while cold cloths are placed on the forehead. The room should be kept dark and quiet, as there is always great sensitiveness to bright light and noises. Although phenacetine and antipyrine afford relief for a time, they tend to lose their effect, and the attacks appear to become more frequent if large quantities of these drugs be taken.

(*b*) *In the intervals.*—Overwork and indiscretions of diet must be avoided. Any errors of refraction should be looked for and corrected. No drug has any specific effect in warding off the paroxysms. If anæmia be present iron should be given. The prolonged administration of bromides has proved beneficial in some instances, and the addition of tincture of gelsemium appears to increase its effect. Among other drugs which may be tried are cannabis indica, nitroglycerine, and caffeine; in some cases a course of iron and strychnine is of benefit. Care must be taken to secure the regular daily action of the bowels.

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IDIOPATHIC EPILEPSY

A chronic affection of the nervous system characterised by the occurrence of repeated attacks of unconsciousness, usually attended with convulsion, and by a tendency to mental deterioration. When the attacks are accompanied by convulsion they are spoken of as *major* fits (grand mal), while momentary attacks without convulsion are described as *minor* fits (petit mal).

Etiology—*Age.*—Out of the 1450 cases tabulated by Gowers, nearly one-third commenced before the age of ten, and three-fourths before the age of twenty. When general epileptic convulsions occur for the first time in a patient over thirty, the probability of some organic disease, such as cerebral tumour, gumma, general paralysis, or of alcoholism should be remembered.

Sex.—The incidence of the disease is almost equal upon the two sexes if all cases of epilepsy are considered together, females preponderating slightly over males in the proportion 21 : 20. In childhood the sexes are almost equally affected, males preponderating slightly. In the decade following the appearance of puberty, however, females are affected much more frequently than males, the proportion being 120 : 100. This is to be explained by the much more profound effect upon the female nervous system of the establishment of puberty. From middle life onwards, on the other hand, the number of males affected exceeds that of females.

Heredity.—Direct inheritance occurs much less frequently than would be expected, but epileptics usually belong to families in which neuroses, such as migraine, insanity, etc., are prevalent. Alcoholism in the parents is a most important factor in the production of the disease. Congenital syphilis is rarely a cause, although syphilitic lesions may cause epileptiform fits (see later).

Exciting causes. — Although great stress is usually laid on supposed causes by relatives, the causes assigned are as a rule of little importance except as determining the first occurrence of fits in a predisposed subject. Frights and falls are the most common exciting causes, but even in these cases a history of minor fits for a long time previously can often be obtained. Reflex irritation from a tight prepuce, or from dentition, are often supposed to be the cause, but the fits are rarely permanently cured by relief of the local condition, and genuine cases of "reflex epilepsy" are very rare. Errors of refraction have been alleged as a cause, but their

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correction does not lead to the cure of epilepsy. Masturbation is common among young epileptics, but is probably a consequence rather than a cause of the disease. Epilepsy often follows some acute specific disease, such as scarlet fever, without any evidence of cerebral embolism or thrombosis.

MAJOR FITS

Symptoms.—Before describing the symptoms, a brief description of a typical fit will be useful. The patient is suddenly conscious of some sensation (aura) which he knows by experience to be the invariable precursor of the fit. Almost immediately he loses consciousness and falls as if shot, often uttering a loud cry. He becomes quite rigid (tonic stage) with his head and eyes turned to one side, the whole body, including the respiratory muscles, being in a state of tonic spasm. In consequence of the arrest of respiration he becomes cyanosed, the veins of the face and neck being greatly distended, and the pupils become widely dilated. Just as he appears to be on the point of asphyxia, the tonic spasm gives way to clonic spasm, and jerking movements, often violent, of all the voluntary muscles of the body come on. Air now enters and is blown out in short puffs, churning up the saliva into foam, which is often stained with blood, owing to the tongue being bitten by the clonic spasm of the jaws. At the same time the face resumes its normal colour. The jerking, which is at first at the rate of about three a second, gradually becomes slower, though not less vigorous, and the last jerk of all is often extremely violent. The patient then lies in a state of coma, with his eyes open and congested, and limbs relaxed, breathing stertorously. The patient is often found to have evacuated his bladder, and occasionally his bowel, during the fit. After the coma has lasted for ten minutes, or longer, he recovers consciousness, with no recollection of what has taken place, or the coma may pass insensibly into a natural sleep, from which he wakes after a few hours. It must be remembered, however, that no two cases of epilepsy are alike. The loss of consciousness and the occurrence of a convulsion are invariable in major fits, although the latter is extremely variable in severity, not only in different individuals, but in the same patient, it being at times so slight as to escape notice. The other phenomena, the aura, the initial cry, the passing of urine, etc., are so variable that their absence cannot be taken as negative evidence in diagnosing the case. But where they are present they form very strong evidence of the epileptic

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nature of the convulsion. We may consider now the symptoms in detail.

The aura.—There is a definite aura in only about half the cases, and in those patients who experience one it does not precede all their fits. It is very important to obtain an accurate account of the aura, as, if the epilepsy should be due to a local lesion, it may be of great value in localising it. The aura may be—*Motor*, consisting in twitching of some part of the body, such as the thumb, slow contraction of the limbs, or rotation of the head. In idiopathic epilepsy a motor aura is usually very brief, in contrast to the deliberate, characteristic onset of "cortical fits." *Sensory.*—(*a*) Common sensation—curious sensations of numbness, or "pins and needles," in almost any part of the body. (*b*) Visceral sensations, usually epigastric, hypogastric, or præcordial. (*c*) Special sense auræ are rare, and consist of flashes of light or moving balls of fire, often coloured; sudden reports or hissing noises; or, very infrequently, crude sensations of taste or smell. (*d*) Vertigo (see p. 294). (*e*) Psychical auræ, *e.g.* indefinable terror, some vision which occurs before every fit, or, in some cases, a complicated dream, the details of which are often forgotten, although the patient knows that he always has the same dream ("dreamy state," "reminiscence").

An *epileptic convulsion*, as already stated, occurs in two stages. It commences with a *tonic stage*, often ushered in by a little twitching, most often in the thumb or face. The patient falls, with or without a *cry*.

This "epileptic cry" occurs in the majority of severe attacks. It is produced subsequently to the loss of consciousness by tonic spasm in the laryngeal muscles and the respiratory muscles, air being forced through the spasmodically closed glottis, by the powerful action of the respiratory muscles as they become involved in the spasm.

The head and eyes are turned to that side on which the convulsion is stronger, and the mouth is usually also drawn to the same side. The arms are usually partially raised, the elbows and wrists being held rigidly flexed. The fingers are sometimes flexed, sometimes extended. The legs are almost always rigidly extended. During this stage dislocation of the shoulder or jaw may occur. The chest is held rigid by the contracted respiratory muscles. During the tonic stage the pupils are dilated and insensitive to light, and the conjunctival reflex is lost. This stage is not of long duration, and after a few seconds it is succeeded by the *clonic stage*,

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which varies in severity and duration, and lasts sometimes ten or more minutes. Some air now enters the chest, and the alarming cyanosis passes off. The rapidity of the clonus gradually diminishes, but the final shock-like contractions, though slow, are often very violent. It is during this stage that the tongue is bitten by being protruded and caught.

Post-convulsive phenomena.—The convulsion is succeeded by coma. This may be momentary, or may be deep and prolonged for hours. Abrupt recovery of consciousness is rare, the patient usually remaining drowsy for some time, or the coma may pass insensibly into normal sleep. On waking there is complete oblivion of what has taken place. In not a few cases, before there is any return of consciousness the patient may get up and perform complicated and apparently purposive acts. This condition is known as *post-epileptic automatism.* The most common acts are attempting to undress, running out of the house, and even maniacal violence. The medico-legal importance of recognising this condition is obvious, and in this connection it must be borne in mind that, while no remembrance of the automatism exists after the attack, yet memory of past events is present during the automatic state, and patients may commit criminal actions of the most complicated character against people they dislike, with much apparent malice aforethought.

Psychical disturbances usually succeed attacks of minor epilepsy, and it is rare for active mental disturbance to follow an attack of severe convulsions. The most severe maniacal outbursts may occur after a minor seizure so slight as to be almost unnoticeable, and for this reason may be apparently spontaneous, and the same may be said also of post-epileptic hysteroid attacks.

Vomiting sometimes occurs at the end of the clonic stage, and before the patient has recovered consciousness, and, unless the patient is turned from his back on to his side, food may enter the larynx and suffocation result.

The knee-jerks are commonly lost for a few seconds after the cessation of the fit, but this is soon succeeded by an increase which persists for some minutes. After ordinary epileptic fits there is general prostration, which varies in amount, and there is often a feeling of muscular fatigue owing to the previous spasm. This is *general*, while *local* paralysis following a seizure is characteristic of cortical or epileptiform fits. In rare cases there is abrupt recovery, and the patient seems and feels none the worse for the attack.

Urine is usually excreted abundantly after the fit, and has been

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found to be more toxic when injected into animals than that secreted before the attack. Occasionally it contains a small quantity of sugar or albumen.

In some instances the clonic stage is at once succeeded by another fit. This may be continued for some hours, and is then spoken of as the "status epilepticus." During the status the temperature rises even to hyperpyrexia, and there is great danger of death from exhaustion.

MINOR FITS

As has already been mentioned, all epileptic seizures are not accompanied by convulsion. Some epileptics have only these minor attacks, but as a rule both are exhibited in the same person. They are usually spoken of by patients as "sensations," "faints," "giddy turns," etc., and their epileptic nature is often unsuspected.

The forms of these attacks are very numerous. There is almost always brief loss of consciousness, so brief that the patient may not fall; sometimes it is only impaired. The loss of consciousness is often preceded by some warning sensation similar in character to the aura, which has been described in connection with the major seizures. If a patient is the subject of both major and minor fits the aura is usually the same in both, but in not a few epileptics there is a different aura for each kind of fit. Amongst the common forms of minor attacks are momentary pallor of the face with fixed staring expression and blinking of the eyes, sudden lapses in action and conversation, sudden starting, dropping anything that is being held, sudden falling, fainting, and strange feelings. Urine is occasionally passed in minor attacks, and nocturnal enuresis may be the only sign of attacks, of petit mal that occur at night. Post-epileptic automatism, mental excitement, and hysteroid convulsions are more common than after major attacks, and these symptoms are in some cases the only indications of an attack of petit mal having occurred.

A considerable number of the sufferers from epilepsy have in infancy suffered with infantile convulsions of considerable severity. Sometimes these convulsions, commencing in the early part of the second year, are continued into later childhood and adult age as established epilepsy. Again, in other cases the infantile convulsions, after recurring during several months, cease, and only on reaching late childhood or puberty do fits make their reappearance. In many of such cases, however, during the interval between the

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infantile convulsions and the occurrence of epileptic fits, attacks of minor epilepsy occur which are often scarcely noticed by those who have care of the child. Whether the interval between the occurrence of infantile convulsions and the later occurrence of epilepsy be bridged over by attacks of petit mal or not, it is probable that the functional instability of the cerebral cortex which leads to the occurrence of the convulsions in infancy persists as the passive cause of the epilepsy of later years. And since it is to the general malnutrition represented by rickets that infantile convulsions owe their chief origin, many cases of epilepsy must accordingly be attributed to this disease.

Dangers of epileptic attacks.—Epilepsy is often indirectly the cause of death, but, except in the case of the status epilepticus, there is little danger from the convulsion itself. But injury or death may be caused by the patient falling into the fire, against machinery, or among street traffic. His occupation should, therefore, be chosen that he may be as little as possible exposed to dangers of this kind. Danger to life also comes from the occasional vomiting before the patient has recovered consciousness, and from an occasional tendency for him to turn over on his face after a fit with a risk of suffocation by his pillow. The first is to be guarded against by turning him on to his side if he begins to vomit, and the second by making him sleep on a porous horse-hair pillow, through which he can easily breathe.

Mental state of epileptics. — Although occasionally epileptics have been met with who showed exceptional mental endowments unimpaired, there is, as a rule, considerable deterioration. Defective memory for recent events, irritability of temper, incapacity for initiating any work or duty without help or encouragement from others, and often downright laziness, are almost always met with in confirmed epileptics. But in many there is more pronounced mental change, taking the form of gradually progressive dementia, with occasional periods of excitement. Epileptics form a considerable proportion of the cases in our asylums, and explosive maniacal attacks often replace the convulsions.

Pathology. — The structural changes in the brain which underlie epileptic phenomena are quite unknown. The changes which have been described—slight opacity and thickening in the membranes, and induration of the cornu ammonis—are generally recognised to be secondary. Both clinical and pathological evidence proves beyond doubt that the seat of disorder in epilepsy is the cortex of the cerebral hemispheres. Organic epilepsy is

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found always to be due to some lesion of the cerebral cortex (see cortical fits). Electrical stimulation of the cortex in animals produces convulsions which, according to its degree, are local or general. The occurrence of highly complex psychical and special sense auræ in epilepsy, and its association with the peculiar disorder of consciousness, automatism, point to the involvement of the highest nerve centres, while the movements that occur at the commencement of the convulsion, if deliberate, are such as we know to be differentiated only in the cortical centre. Important, in this connection, are cases recorded and cited by Sir William Gowers, in which the occurrence of hemiplegia due to a lesion in the region of the basal ganglia in cases of general epilepsy caused permanent arrest of the convulsion in the opposite half of the body. The exact regions of the cerebral cortex concerned are far from being proved. It has been shown that convulsions may be produced by excitation of any part of the cortex, including those concerned with the special senses, and it has been presumed that where visual auræ are present in the attacks the occipital lobe is the *locus morbi*, the uncinate gyrus with auræ of taste and smell, the temporo-sphenoidal with auditory auræ, etc. In attacks commencing bilaterally with rapid loss of consciousness and absence of aura Dr. Hughlings Jackson suggests the prefrontal lobe as the seat of the disease.

The muscular spasm which constitutes the most conspicuous feature of the epileptic attack and the sensations which the patient experiences before losing consciousness must be regarded as due to the sudden violent action, the "discharge" of the cortical gray matter. The suddenness of this discharge in epilepsy does not transcend that of normal action in a degree that excludes the reference of both phenomena to the same process. The former, however, is an irregular discharge in the absence of the normal stimulus. As a result of each spontaneous discharge the affected elements become possessed with a difference in physiological constitution which makes the occurrence of similar discharge more easy, and this in a degree proportioned to the frequence of its occurrence. It is this tendency that renders such a disease as epilepsy self-perpetuating, and for this reason it is only by the long-continued restraint of the morbid process that normal stability of the affected nerve elements and cessation of spontaneous discharge can be obtained.

Diagnosis.—When there is a history of liability to convulsions, the diagnosis has to be made, in the case of major fits, from hysteroid

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convulsions (p. 327) and malingering; and in the case of minor attacks from fainting and vertigo (p. 295). Major fits also have to be distinguished from convulsions due to local disease of the brain, such as tumours, and from uræmic convulsions and convulsions due to drugs.

A medical man is unlikely to be imposed on by a malingerer if he have an opportunity of witnessing one of the fits, and the preservation of the pupil reflex will further aid in distinguishing. From hysteroid attacks the character of the convulsion, tonic followed by clonic stage with groaning but no speech in epilepsy, rigidity with opisthotonos, followed by struggling purposive movements, and shouting or screaming during the fit in hysteroid convulsions, will usually enable a diagnosis to be made. But it must be borne in mind that hysteroid convulsions are often post-epileptic phenomena. From fits due to lesions of the cortex the diagnosis is by no means easy in all cases, as in the early stages the fits from a tumour may be general. Usually, however, they are one-sided, and are characterised by definite aura, slow march of the spasm, late loss of consciousness (or consciousness may be preserved), and, most important of all, paralysis of the part in which convulsion has occurred for some time after the fit. Optic neuritis is present in many cases of cerebral tumour, and should be looked for in every patient who comes for treatment of convulsions. Uræmic convulsions will be of recent origin, and the other signs of renal disease should prevent a false diagnosis. Convulsions may occur from time to time in those who have had some head injury or local lesion years before. Convulsions may occur temporarily as a result of an alcoholic bout.

The **prognosis** in epilepsy involves several separate questions —(1) the danger to life; (2) the prospect of a spontaneous termination of the disease; (3) the prospect that by treatment the disease may be cured or the attacks arrested.

The chief danger to life is from the exposure to accident which the attacks involve. Death from asphyxia may occur from choking if an attack should occur during a meal or if a patient vomit after the attack while still comatose. Another danger is the tendency of patients to turn on the face after an attack in bed, when suffocation may occur in the post-epileptic insensibility. Falling into the fire or on to machinery are not uncommonly the causes of death in epileptics, but by far the most common accident is drowning. The danger of such accidental death is, however, very small. Death from the severity of a single fit is very rare, and the occurrence of

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status epilepticus, often fatal, is so uncommon that the accompanying danger to life may be neglected in considering prognosis.

Spontaneous cessation of the attacks is not very uncommon, occurring sometimes after the age of twenty, and still more frequently as life advances; but it is too rare an event to be reasonably anticipated in any given case. The expectation firmly rooted in the popular mind that attacks in girls cease with the first menstruation is not justified by facts. Marriage only influences the course of the disease, in that pregnancy in many cases causes temporary cessation of the fits, which, however, return after delivery.

As regards arrest and cure by treatment, the prognosis is slightly more favourable in females, and distinctly more so if the disease begins after the twentieth year than if it commence between ten and twenty. It is better the shorter the duration of the disease and when the disease is inherited than when no heredity can be traced. This remarkable fact, first pointed out by Herpin and conclusively proved by Sir William Gowers, is also true as regards some forms of insanity. It is better the longer the interval between the attacks and worst when they occur almost daily. It is better if the attacks occur in the sleeping or in the waking state only, than if they occur in both. Again, the prognosis is more favourable if there is no considerable mental impairment, and if all the attacks are of the severe variety, than if there are minor seizures, and better if the attacks are preceded by an aura than if they occur without any warning. A really good prognosis can seldom be given unless the attacks are arrested by bromide in moderate doses, such as sixty grains a day, and when it is possible to secure persistence of arrest with effective treatment for a sufficient time, two or three years.

Treatment—*General.*—Attention to the general health is most important. Regular daily action of the bowels must be secured, and the patients should live as much in the open air as possible. They must avoid situations of danger, such as would be caused by working among machinery, on scaffolds, etc. They must not be allowed to bathe. Their diet must be plain, but abundant, and it is inadvisable for butcher's meat to be taken more than once a day. As a general rule stimulants should not be permitted. It is highly desirable that some occupation should be found for them, as mental degeneration occurs more readily when they are left in idleness. Farm or garden work are most suitable forms of occupation. Work involving close application indoors, or much responsibility, is often injurious, and study under conditions of strain, as for examinations, should not be permitted.

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Drug treatment.—Bromide of potassium, or the bromides of sodium, ammonium, or strontium have by far the greatest influence. If treatment is begun early, and kept up continuously, complete cessation of the fits occurs in about ten per cent of cases, and even in chronic cases it greatly diminishes their frequency. If the fits are only occasional half a drachm may be given in water every night. If they are more frequent, fifteen to thirty grains may be given three times a day after meals. The medicine must be taken with absolute regularity, and at least one daily dose should be taken for two years after the occurrence of the last fit.

In many persons the administration of bromides gives rise to acne. This is greatly lessened by adding three or four drops of liquor arsenicalis to each dose, but the tendency for arsenic to cause neuritis and pigmentation of the skin of the trunk must be borne in mind. When the disease does not yield to bromides, digitalis, belladonna, or borax, may be added to the mixture, along with cascara or other aperient. If the patient is losing flesh, cod-liver oil should be given. The administration of bromide is often thought to be responsible for the mental deterioration which occurs, but it takes place at least as rapidly in those who are treated by other drugs. Surgical treatment by simple trephining, by excision of the part of the brain supposed to be affected, or by excision of the sympathetic, has been at different times recommended, but the results have been unsatisfactory.

Treatment of the attack—Arrest.—When fits are ushered in by a deliberate warning the attacks may sometimes be cut short at the onset. In cases in which the attack begins unilaterally in the hand or foot the application of a ligature round the limb or forcible extension of the contracting muscles is sometimes effective. A more permanent result is sometimes produced by a circular blister about an inch broad applied round the limb. Where the warning is general a powerful mental effort, or some exertion, such as walking quickly, or some powerful sensory stimulus, such as the swallowing of a teaspoonful of common salt or the smelling of strong ammonia, will sometimes arrest the fit. A whiff of amyl nitrite has sometimes the same effect.

Treatment during the attack.—In a major attack assistance is first called for to prevent biting of the tongue, and for this purpose the handle of a spoon, a tongue depressor, or, still better, a thick piece of indiarubber should be forced between the teeth. The patient should be placed in the supine position and the clothes round the neck loosened. Little or nothing can be done to arrest the developed

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attack. Where nocturnal attacks occur it is advisable that the patient should sleep upon a hard bed and use a hard pillow, for the result of convulsion is generally to turn the subject upon his face and asphyxia may result in the stage of post-epileptic coma. Not a few epileptics lose their lives in this way, and Sir William Gowers has stated that if a person be found dead in bed lying upon his face there is the strongest presumptive evidence of death after convulsion. When vomiting occurs during the attack the patient should be turned on to his side and the mouth opened and cleared, so that the vomited material is not drawn into the larynx, for this has occasioned death on not a few occasions. The sleep subsequent to the attack should remain undisturbed, since patients when roused have much more headache than if they are left.

In the status epilepticus bromide often fails to do good. As the repeated attacks cause profound exhaustion attention should be paid to the administration of liquid nourishment and stimulants by the nasal tube. Strychnine is useful hypodermically. By far the most useful drug in arresting the convulsions is hyoscine, which should be given as the hydrobromate hypodermically in doses from $\frac{1}{200}$ to $\frac{1}{100}$ grains (Gowers). Morphia hypodermically is also useful in doses of $\frac{1}{4}$ to $\frac{1}{2}$ grain, and chloral gr. xv. every three hours is sometimes efficacious. The inhalation of chloroform is useful to produce temporary cessation of the convulsion, but it rarely has any permanent effect.

In children petit mal sometimes takes the form of a sudden fall, the head invariably coming in contact with the ground first. Much suffering and much unsightly scarring of the forehead, as well as the deleterious effects of the repeated concussion, may be obviated by the patient wearing continuously a cap with a thick inflated india-rubber margin resembling a bicycle tyre.

CORTICAL FITS

Syn. Local, Jacksonian, or Epileptiform Fits

These terms are applied to certain fits which are associated with disease of the cerebral cortex or the meninges in the "motor region." But it must be remembered that cortical lesions, even in the motor area, may give rise to general convulsions exactly like

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those described in idiopathic epilepsy. This is probably dependent upon the rate of the discharge and the rapidity with which it spreads. When the discharge is slow and the spreading deliberate, the typical cortical fit occurs. Where the discharge is rapid and the spread so quick that the whole of both cortices are almost instantaneously affected, a general convulsion results. In rare cases convulsion of local onset and slow spreading occurs in idiopathic epilepsy.

Etiology.—Cortical fits are seen in their most typical forms in cases of tumour or abscess involving the cortical or subcortical portions of the parietal region, in the second stage of general paralysis of the insane, in cases of meningitis, and after local injury. They may occur as a consequence of any local cortical lesion, and in some cases of uræmia.

Symptoms.—The symptoms, although bearing a general resemblance to those of epilepsy, present some important differences.

(*a*) Aura. There is usually a well-marked aura, consisting either of twitching in some part or of local sensory phenomena. This may last for a few minutes or longer before the actual convulsion commences.

(*b*) Convulsion, first tonic and then clonic, begins in the part where there has been twitching and spreads deliberately to other parts of the same side. Thus, if the fit began in the thumb the march of the spasm would be: wrist, elbow, shoulder, hip, knee, foot, in every successive fit. This *deliberate march* is very characteristic. The convulsion is nearly always unilateral, but in the more severe attacks the fit may end by becoming general.

(*c*) Consciousness is nearly always preserved during a large part of the fit and may be retained throughout. The convulsion is rarely painful.

(*d*) Subsequent local paralysis. After the fit the side affected is usually found to be paralysed. This paralysis gradually clears off, remaining longest in the part in which the convulsion started, and may not have completely cleared off before another attack occurs. In many cases there is already some local paralysis before the fits occur.

SENSORY FITS.—As local lesions in those parts of the cerebral cortex concerned with movements of the body may give rise to the local convulsion and symptoms above described, so may a lesion of those elements of the cortex which are concerned with the registration of sensation give rise to a sensory disturbance in every way analogous to the motor cortical fit; for an instance, a curious

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sensation commencing in the index finger, spreading to the hand, forearm, arm, shoulder, and face successively, and then running down one side of the body to the foot. Such sensory disturbances may occur alone without any motor complication, and are of the same weighty importance as are the local convulsions in the diagnosis and localisation of disease. They may, however, precede local convulsion as its aura, or the two may proceed *pari passu*.

Lesion of the cortex in the regions concerned with the special senses may produce similar visual and auditory hallucinations and olfactory and gustatory sensations.

In cases of gross organic cortical disease, such as tumour, the accurate observation of the cortical fits affords the most valuable aid in the localisation of the lesion, since the part in which the initial spasm occurs and the part in which paralysis persists longest are those innervated from the parts of the motor cortex in which the irritative lesion is situated.

The **treatment** varies with the cause. In cases of injury, abscess, or tumour, surgical intervention may be desirable. If there is any reason to suspect a syphilitic origin, iodide of potassium and mercury should be energetically administered. Improvement of symptoms under such treatment often occurs where a new growth, not syphilitic, is present; this may be so marked soon after the commencement of treatment that false hopes may be entertained, but it is temporary only, the symptoms becoming again aggravated notwithstanding the continuance of therapeutic measures. Bromide of potassium should also be given as for idiopathic epilepsy.

INFANTILE CONVULSIONS

Convulsions closely resembling those of epilepsy are common in infancy, in which the reflex excitability of the nervous system is much greater than in later years. At the time of birth the nervous system, especially the nervous structures of higher function, are incompletely developed, and functional capacity, with its most important element—stability—is only fully acquired when structural development becomes complete. Consequently, until such structural perfection is reached the nervous system is unstable, and is prone to respond to peripheral disturbance by disorderly liberation of energy, which may appear as convulsion, spasm, or increased reflex

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excitability. Statistics compiled by Drs. Coutts and Gossage show that an inherited neurotic tendency is a most important predisposing factor in the production of infantile convulsions.

Rickets is by far the most important predisposing cause. This constitutional condition, affecting as it does every system of the body, retards the development and diminishes the stability of the nervous system, while the disordered function of other parts of the body frequently serve as potent causes for the setting up of reflex convulsion (see Rickets, Vol. II. p. 250). The exciting causes of convulsions in a rickety child are most commonly intestinal disturbances, especially those resulting from indigestible food, and difficulty in cutting the teeth. The latter cause has given rise to the familiar term "teething convulsions." Often, however, some peripheral impression, frequently trifling in nature, may cause convulsions, and in many cases no cause can be discovered. Intestinal parasites are often regarded as the excitant. Round-worms sometimes cause reflex convulsions, but thread-worms never, while tape-worms are very rare in children.

Infantile convulsions are also common at the onset of acute diseases, especially of pneumonia; and it may be said that where rigor occurs in the adult, convulsion occurs in the child. Similarly the profound nutritional effect of congenital syphilis upon the system is a potent cause of convulsions.

Much more rarely convulsions are symptomatic of cerebral disease, occurring sometimes at the onset of infantile hemiplegia and of cerebral diplegia; they may also be the first symptoms of meningitis. Conditions of asphyxia are potent in exciting convulsion in children, and these may be met with in the asphyxial stage of a severe whooping-cough paroxysm, in laryngismus stridulus, in the dyspnœa of diphtheria and pneumonia, and in congenital morbus cordis where cyanosis is present, or even at the end of a violent paroxysm of screaming. The convulsion commonly seen in children "in articulo mortis" is probably of this nature.

Uræmic convulsion very rarely occurs in children, and may be indistinguishable from convulsion due to other causes. It is probable that such cases are the results of scarlatinal nephritis. The presence of albumen in the urine of a child suffering from recurring convulsions should always be excluded before a good prognosis is given.

The period of infancy during which convulsions occur most frequently coincides with the time of incidence of rickets, and is between the sixth and eighteenth month of life.

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As has been above stated, a neuropathic heredity, especially of epilepsy, is a predisposing cause, but by far the most important relation of infantile convulsions to epilepsy is the tendency for the former to recur and to persist as an established epilepsy. A large proportion of cases of idiopathic epilepsy originate from infantile convulsions due to rickets.

Symptoms.—The convulsions resemble those of true epilepsy, but are less violent. They consist of a tonic followed by a clonic stage, but both tonic and clonic stage may exist alone. The convulsion is nearly always bilateral, but often does not affect the whole body. The convulsion often lasts longer in each attack than in the epilepsy of older subjects, and, in the slighter tonic forms, an attack may last half an hour or more. Sometimes the convulsions follow one another rapidly, and recovery in the intervals may be imperfect.

The onset is usually sudden, but there may be prodromal symptoms, such as restlessness, irritability, twitching of the face and abnormal movements of the eyes.

The convulsions of rickets may be few, and cease after a few days, or they may recur. Not infrequently they occur at intervals during the second year of life, and then cease. If, however, they continue for a longer period than this the condition must be regarded as epilepsy, since it continues after its cause has ceased.

Associated with the convulsions of rickets are the peculiar conditions—laryngismus stridulus, facial irritability, and carpopedal contractions.

LARYNGISMUS STRIDULUS consists of spasm involving the laryngeal and other respiratory muscles. It is a local form of convulsion, and arises from the same exciting causes as do the general convulsions. After being startled or annoyed the child holds his breath and becomes cyanosed, the head is thrown back, the neck forward, the eyes are staring, and the pupils contracted. The countenance bears an expression of extreme anxiety, the face perspires, and the vessels of the neck are swollen. The glottic spasm lasts usually from fifteen seconds to two minutes, but the glottis may remain closed till consciousness is lost or even till death occur. When the spasm relaxes the child draws in his breath with a crowing sound which is often repeated with a few subsequent inspirations, after which the child often seems quite well again and is ready for play.

This condition may occur as late as the ninth year; it is most common in the second year. It not infrequently is first noticed

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after measles or whooping-cough in subjects otherwise predisposed. Facial irritability always accompanies laryngismus.

FACIAL IRRITABILITY (Chvostek's sign).—This condition, which is frequent in rickety children and occurs invariably in tetany, consists in a remarkable increase in excitability of the motor nerves and muscles to direct stimulation. It is elicited by tapping the face over the pes anserinus of the facial nerve, when an immediate contraction of all the muscles supplied by the facial nerve occurs.

CARPOPEDAL CONTRACTIONS (see Tetany).

Treatment.—The immediate treatment of the convulsion is directed towards its arrest and the subsequent treatment to the removal of the cause and to prevent the repetition of the convulsion.

The convulsed child may be placed at once in a warm bath at a temperature 95° to 98° F., but if this has no immediate effect it is useless to repeat it. If the convulsion be severe chloroform inhalation is called for, even though there is profound unconsciousness; the spasm rapidly ceases and consciousness usually returns with the passing off of the anæsthetic. The most useful remedy is the rectal injection of chloral and potassium bromide in doses of three grains of each to an infant of six months of age. Inhalation of amyl nitrite has been recommended, but it is not so efficacious as the administration of chloroform.

The attack being over, a mercurial purge should be given and bromide of potassium administered in doses of three grains to a child under six months; five grains to one between six and sixteen months. Two or three doses should be given each day and continued for about a week. As the bromides require some time for the development of their physiological action, the convulsion may return before this has expired and should be combated with chloroform inhalation.

Any cause for the convulsion should be sought for and treated. The treatment appropriate for rickets and congenital syphilis must be carried out where these diseases are manifested. Any possible source for peripheral irritation should be looked for and removed.

The greatest care must be bestowed upon diet, ventilation of rooms, fresh air, sunlight, etc.

Laryngismus stridulus is most efficiently treated with bromides and chloral. Where the paroxysms are so prolonged as to require immediate treatment the child may be immersed in a warm bath, and cold water may be dashed over the face. Ammonia or chloroform held to the nostrils sometimes causes the spasm to abate. If

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life should have become apparently extinct, artificial respiration should be resorted to.

In all cases of convulsion, etc., where rickets is present the most important treatment is the general management of the child, which is described under that disease (Vol. II. p. 262).

TETANY

This term is applied to a condition characterised by the occurrence of muscular spasm, chiefly in the peripheral part of the extremities, associated with peculiar excitability of both nerve and muscle to electrical and mechanical stimuli.

Etiology.—Though most common in children, it may occur at any period of life. In infancy males suffer far more commonly than do females, while in adult life the reverse is observed. In infantile tetany rickets is almost invariably present, and it is probable that the two conditions are here simultaneous effects of one cause. Sometimes it is associated with exhausting diarrhœa. In the adult it is met with in connection with several different conditions: (1) in connection with pregnancy and prolonged and exhausting lactation, and it may recur with successive pregnancies and lactations; (2) associated with gastric dilatation, where it is liable to follow the operation of lavage, prolonged gastro-enteritis and the presence of intestinal parasites; (3) following extirpation and, rarely, disease of the thyroid gland (tetanilla strumipriva); (4) after specific fevers, especially enteric fever; (5) it has been known to occur as an epidemic disease.

Symptoms. — The muscular spasm commences somewhat abruptly, and affects firstly both hands, later both feet, and may spread subsequently to involve the proximal portions of the limbs and even the trunk and face. For a few hours, or even days, before the spasm appears, tingling, numbness, or burning sensations usually are felt in the periphery of the limbs. In some cases the spasm commences in the feet or trunk and has an irregular march. The attitude assumed by the limbs during the spasm is characteristic. In the upper extremity the thumb is adducted and flexed into the palm beneath the other fingers, which are flexed at the metacarpo-phalangeal joints and extended at the other joints. The palm is hollowed, and the hand, from its conical shape, has been called the "accoucheur's hand." In rarer cases flexion of the fingers at all

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joints, or the position known as "main en griffe" may occur. The wrist is flexed and deviates somewhat to the ulnar side; the elbow is flexed and the shoulder adducted, the forearm being pronated and carried across the chest, being held a little in front of the trunk. In the lower extremities the toes are strongly flexed, especially the hallux, and adducted; the foot is arched and inverted and the ankle extended. When the trunk is affected there is usually emprosthotonos, rarely opisthotonos. There is, in severe cases, some spasm of the tongue and laryngeal muscles, and sometimes of the respiratory muscles. The term "carpo-pedal contraction" has been applied to the infantile cases usually associated with rickets, where spasm is confined to the hands and feet. (See Rickets, vol. II. p. 259.)

Movement is interfered with in proportion to the severity of the spasm, and where this is severe voluntary movement is impossible.

The spasm is paroxysmal and, after lasting a few minutes to a few days, passes off slowly, often incompletely, to recur again. Pain in any degree, from a feeling of slight cramp to most severe pain, may accompany the spasm. Passive movement is as a rule very painful. Increased excitability of both muscle and nerve to percussion is a characteristic symptom and is known as Chvostek's sign; during the intervals between the spasms percussion of the muscle or of its motor nerve at once elicts a marked contraction. A similar condition in the facial muscles occurs sometimes in rickets apart from tetany. Compression of a limb so as to impede the circulation, or pressure on the nerve trunks, at once induces a spasm, this is known as "Trousseau's phenomenon."

The electrical excitability of both nerves and muscles to both faradism and galvanism is increased, and often the anodic opening contraction occurs before the kathodic closing contraction, and with it a tetanus resembling the "Ritter's tetanus" which occurs experimentally in fatigued muscles. This peculiarity was first pointed out by Erb, and is known as "Erb's tetany reaction."

The reflexes are not materially altered, but their manifestation may be interfered with by the spasm.

The attacks in infants are often associated with pyrexia; there is sometimes a hard œdema of the extremities present.

Prognosis. — Tetany usually ends favourably, except when occurring after thyroidectomy and in connection with gastric dilatation. In weakly children diarrhœa and respiratory complications are responsible for most of the fatal results. Tetany in pregnancy usually ceases with delivery; that of lactation with the weaning of

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the child. Tetany is always prone to recur in the same subject if again exposed to the influences known to be causal factors of the disease.

Pathology.—No definite lesions have been found. In one case which one of the writers (W. S. C.) examined by modern methods no microscopic change could be detected. The seat of the disease is a matter of speculation, the peripheral nerves, anterior horn cells of the spinal cord, cerebellum and cerebral cortex, being each supported by different authorities as the *locus morbi*. There is certainly grave alteration in the nutrition of the peripheral nerves and the actual spasms are probably set up reflexly. There is great probability that some poison of gastro-intestinal origin, circulating in the blood, is responsible for the symptoms, but of its nature we are ignorant.

The **treatment** of tetany consists in the removal of the cause, where possible, as in rickets, lactation, etc. Where gastro-intestinal troubles are present their treatment by intestinal antiseptics and careful feeding is indicated.

Thyroid feeding in some form is essential in those cases in which the thyroid body appears to be at fault. All-important is the improvement of the general health and nutrition.

For the palliation of the spasm the patient should be kept at rest and in an equable temperature. Stimulating liniments and the application of cold lessen the pain. Chloral and bromid s are the drugs most efficacious in lessening the spasm.

HYSTERIA

This term is applied to a peculiar cerebral and psychical state dependent on some disturbance of the highest nervous centres, but usually showing itself in some functional derangement of the lower cerebral or spinal centres without any recognisable structural change. Hence occurs a long and varied train of symptoms in hysterical patients: such as mental symptoms and alteration of disposition from the disorder of the highest centres, convulsions, visual and other sensory symptoms due to functional cerebral derangements, or paraplegia from interference with the functions of the spinal cord.

The term hysteria is often misapplied. Its derivation from the Greek word signifying the womb, indicates the old and erroneous

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idea that the condition was due to uterine disorders, while others use the term as synonymous with shamming. As this error is common among the laity, the medical man should be careful in using the word to explain that he does not make any implication of malingering. The condition is a real one, and much unintentional cruelty resulted in those days from treating hysterical women as semi-criminals in the spirit of the Middle Ages, when they were exposed to most severe treatment in order "to drive the devil out of them." Further confusion has arisen owing to the fact, which is being tardily recognised as a result of Dr. Buzzard's teaching, that hysteria and definite organic disease of the brain and spinal cord frequently co-exist, and the hysterical symptoms may be so pronounced as to mask the symptoms of organic disease.

The term "functional disease" is now commonly used to indicate the conditions here described, but while its use in this sense is void both of etiological objection and of the unpleasant connotations which the laity attach to the term hysteria, it introduces an arbitrary limitation of meaning; for this term should include all those diseases which, in the present state of our knowledge, are without discovered morbid structural changes, as, for instance, epilepsy, migraine, etc.

Etiology.—Hysteria is not equally common in all countries, and the symptoms exhibited by patients also vary among different races. Convulsive phenomena, for example, appear to have been much more frequent in the Middle Ages, and are still much more prevalent in France than they are in Anglo-Saxon countries. In this country an undue proportion of cases occurs among Jews.

Age and sex.—In rare instances only is hysteria met with in children; it chiefly affects girls between the age of puberty and twenty-five, although symptoms may persist long after that age. The symptoms are sometimes first manifested at the menopause. It is reckoned to be twenty times as common among women as among men, and of males affected the great majority are lads about puberty.

Heredity undoubtedly plays a large part in the production of the nervous state. There is frequently direct inheritance, and in nearly all there is a history of serious brain disease, such as epilepsy or insanity in other members of the family, or of collateral branches. Further, it must always be remembered that faulty training of children, by allowing self-indulgence instead of inculcating restraint and unselfishness, often lays the foundation of hysteria in later life in children with a neurotic inheritance.

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Exciting Causes—*Psychical.*—These are chiefly of an emotional nature, such as a sudden fright, or the sight of some terrible accident, or prolonged anxiety culminating in some emotional shock, such as the death or illness of relatives, financial reverses, or some untoward turn in a love affair.

Physical.—Diseases of the generative organs, usually of a slight degree of severity, are present in many cases. Their influence, however, has certainly been overrated, and beyond the depressing effect on the nervous system of the troubles, it is doubtful whether they have any specific influence in causing hysterical symptoms. Tenderness in the iliac regions is common in hysterical women, and has been supposed to be characteristic, and to indicate ovarian irritation. But this tenderness is almost always to be elicited in women, and tenderness in the same position has often been observed in neurasthenic men. Sexual excess, both in men and women, and masturbation, especially in boys, is a powerful exciting cause of hysteria. It has sometimes been alleged that continence brings about hysteria, but if this ever is the cause, it is exceedingly rare.

Injury may often lead to the development of hysterical symptoms. This is probably due, not to any traumatic lesion of the nervous system, but to the emotional shock associated with the receipt of injury.

Hysterical symptoms have also been frequent in epidemics of lead poisoning due to the contamination of drinking water by minute doses of lead.

Lastly, hysterical symptoms may be brought about, or, if present before, greatly exaggerated by the presence of organic disease, especially of the brain and spinal cord, by fevers, especially influenza and typhoid fever, or by some local disease, such as laryngitis.

Overwork is often assigned as a cause, but is not a very frequent one, overwork being more often responsible for the allied train of symptoms which are comprised under the term "neurasthenia."

All the various causes which have been enumerated will be much more likely to be effective if there be any disturbance of the general health at the time produced by dyspepsia, unhealthy surroundings, etc.

Symptoms.—From what has been said as to the nature of hysteria, it can readily be understood that the symptomatology is a complex tangle. No two cases are alike. In one the psychical symptoms are most obtrusive, in others motor, and so on. There is

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probably no other disease in which the clinical picture presents so much variety. It will be most convenient, therefore, to give a short account of more or less constant features, and then to attempt to enumerate and classify the other symptoms, reminding the student that only a few of these may be present, and that any given case may present any permutation or combination of them.

Patients suffering from hysteria are usually, but not always, more emotional than when in their ordinary health. They are morbidly self-conscious, not only in relation to the outside world, but even more as regards their own emotions and physical sensations. They show a tendency to exaggerate not only their symptoms, but any domestic discomforts they may have, a habit which does not make life more pleasant for them or their families. They love to talk about their symptoms, and show an even greater anxiety than is customary among patients for their case to be considered a remarkable one. This anxiety often leads them actually to simulate some disorder, and it is this not infrequent wilful addition to their symptoms which has led to hysteria and malingering being often put down as synonymous. Among the most common of these *factitious symptoms* are the production of bullæ and other cutaneous lesions by the application of irritants. They are usually only found in positions easily accessible to the patient's right hand, and owing to the anæsthesia, which is often present, the patient is able to produce some very striking results without discomfort to herself. Another common vagary is wilful heating of the thermometer to produce apparent anomalous temperatures, and some voluntarily vomit their food during the day, and surreptitiously obtain refreshment during the night. Others show great ingenuity in concealing their urinary and fæcal excretions, even going to the length of swallowing them, while others add substances, chiefly dyes, to make their urine appear extraordinary. The task of separating real symptoms from those which are simulated is often extremely difficult, but it must not be hastily assumed that because some of the symptoms are factitious the case is merely one of malingering.

In many cases of hysteria some of the symptoms possess characteristics which may be said to be pathognomonic of the disease. (1) They often appear and disappear abruptly from causes which would be trivial in organic disease. (2) They often do not admit of an organic explanation. (3) They are often incongruous. As illustrations of these facts and of the importance of thorough examination in cases of hysteria the following examples may be useful:—A young man of excitable temperament had complete

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paralysis and contracture of the left forearm following a bullet wound in the muscles on the outer side of the forearm two inches below the elbow; the elbow-joint was stiff and there was complete anæsthesia to all forms as high as the shoulder. None of these symptoms were explainable from the nature of the wound, and, though of six months' duration, disappeared after the inhalation of a few whiffs of ether. Another patient with spastic paraplegia was always quite flaccid when his attention was directed from his legs; when asked to bend the knee the quadriceps could be seen to contract at once, and when asked to straighten the knee, which had been passively flexed, the hamstrings at once contracted, no movement in either case resulting. Many patients who, while in bed, are able to perform all movements of the lower limbs vigorously, may be entirely unable to make the attempt to stand, even when supported.

Psychical symptoms.—These are characterised by want of control. Not only are hysterical patients emotional, but they have little control over the expression of the emotions, so that if they begin to weep they are apt to sob loudly, and even to shout and shriek. If they are amused they may laugh with uncontrolled violence. They are often passionate and quarrelsome. In conversation they are exceedingly loquacious. In some cases excitement or depression is so marked that the cases require to be classed as cases of mania or melancholia.

Disorders of speech, etc.—Hysterical mutism is occasionally met with. Hysterical aphasia is seen in rare instances, and is usually of the sensory type (see p. 145). It is extremely difficult to distinguish from that due to tumours in the posterior parietal region.

In this place may be mentioned the so-called hysterical cough, which is better described as a hoarse laryngeal noise frequently repeated. In some cases it is produced by an inspiratory, in others by an expiratory movement. It is a most intractable form of hysteria. Sometimes co-existing with such repeated noises, and sometimes existing alone as a symptom of hysteria, is the habit of phonating during inspiration.

Sensory symptoms.—These vary greatly in different individuals, and even in the same patient at different times. They may be grouped as follows.

Cutaneous anæsthesia, usually affecting sensations of touch, pain, and temperature simultaneously. This in rare instances affects the whole body, but more often there is hemianæsthesia, or, in contrast to other forms, it affects some segment of the body, such as the

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hand, with a definite line of demarcation between the sensitive and insensitive skin. These forms are often spoken of as "glove anæsthesia," stocking anæsthesia, and so on. In other cases there are scattered patches of anæsthesia without any order. The distribution is never that of spinal segments, nerve roots, or nerve trunks, and when half the body is affected the boundary is usually sharply the middle line of the body. Where hemianæsthesia is present it may be complete to all forms, or, while sensibility to touch is only diminished, that to pain may be completely lost. The importance of complete hemianalgesia in the diagnosis of hysteria is great, for it hardly ever occurs in organic hemianæsthesia, and the condition when bilateral (universal analgesia) is unknown in organic disease, except in rare cases of tabes dorsalis and syringomyelia. Sometimes the deeper parts are involved, but usually muscular sense is preserved, so that a hysterical patient whose fingers are absolutely insensitive to a pin can thread a needle and sew. Along with this insensitiveness of the skin there is usually loss of sensation of the mucous membranes. Hence it is usually unexpectedly easy to examine the larynx of a hysterical patient. The anæsthesia may disappear during sleep, the patient quickly drawing the hand away when pricked, and the same reflex movement is seen when the patient is only partially under the influence of an anæsthetic. A most striking peculiarity in hysterical loss of sensibility occurs in some cases who, when blindfolded and tested on various parts of the skin, being told to answer "yes" when they feel and "no" when they do not feel, regularly and quickly answer "yes" when touched upon the sentient skin and "no" when touched upon the anæsthetic part. This phenomenon is certainly not a factitious one, and its occurrence is in accord with certain physiological facts.

Affections of special senses.—These are specially affected along with hemianæsthesia and on the same side. (*a*) Taste is often lost on one side of the tongue. The patient is usually unaware of the loss until it is carefully tested. (*b*) Bilateral loss of smell. (*c*) Deafness. This is usually only revealed by testing, as it is unilateral, and there is increased power of hearing in the opposite ear. The deafness is nerve deafness, bone conduction being unimpaired.

Defects of vision.—Amblyopia is not uncommon, and takes the form known as "crossed amblyopia"; vision of both eyes is here generally diminished, but more in that eye which corresponds with the anæsthetic half of the body; the fields are contracted, that upon the anæsthetic side being most affected, and the fields for colours

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greatly reduced, achromatopsia being often present in the eye most affected without any alteration in the optic disc. Complete amaurosis is rare. Amblyopia comes and goes rapidly, and there are often many attacks. The same characteristics are present in the amblyopia which often precedes signs of optic atrophy in disseminated sclerosis. Hemianopsia or definite scotomata are very uncommon. These are important symptoms, as malingering patients are unlikely to be aware of the possibility of their occurrence.

Hyperæsthesia.—In some cases there is exaltation of the senses. There may be increased sensitiveness of the skin to ordinary stimuli, so that a gentle stroke with a soft brush produces a violent tingling. The special senses also may be affected, and both hearing and vision preternaturally acute, which is often evidenced by the patient's ability to hear conversations carried on in a low tone in another room. Pains in various parts may be complained of, especially a fixed boring pain in the head (hysterical clavus). Infra-mammary pain resembling that sometimes seen in cases of anæmia is not uncommon. Like those of migraine these pains rarely prevent sleep.

A sense of something rising in or constricting the throat and stopping the breath often occurs and is known as *globus hystericus*. A similar phenomenon occurs in some normal persons when under the influence of alarm or emotion. Tenderness on deep pressure over the spinous processes of the vertebræ and in the left hypochondriac and ovarian regions frequently occurs, and when the appearance of symptoms has followed injury the seat of this injury may remain excessively tender. Pressure upon such tender spots may at once excite a hysteroid convulsion, and such spots are termed "hysterogenetic points."

Motor symptoms.—These also vary from time to time. There may be general feebleness or inco-ordination, which in some cases appears to be due to loss of muscular sense. There is one special form, generally met with in young persons, in which, although the patient can move the limbs well when lying down, she is able neither to stand nor walk. To this form the special term "*astasia-abasia*" has been applied.

The motor symptoms may be conveniently divided into paralytic, spasmodic, and convulsive. *Paralytic.*—By far the most common is paraplegia, which is usually of rapid onset, while hemiplegia and monoplegia are about equally frequent and are most frequently of slow onset. The limbs may be either flaccid or rigid, and the loss

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of power may exist in any degree from slight weakness to complete paralysis of limbs and trunk. The nutrition of the limbs is well preserved and the electrical excitability of the muscles is unaltered. The gait, when walking is possible, is often most characteristic, but most difficult of general description. It is a gait which is inconceivable in terms of motor and sensory paralysis, and which suggests that its form is "constructed" in the patient's consciousness. During its performance every difficulty is exaggerated by the patient, and every variety of lurching and hopping movements may be associated. The knee-jerks are greatly exaggerated, and the attempt to elicit them often causes a convulsive shudder of the whole body. Ankle clonus, which is the rule in similar forms of paraplegia due to organic spinal disease, is rarely if ever present in cases of pure hysteria, but a form of clonus is not uncommon which is started by a voluntary movement of plantar extension of the ankle on the part of the patient. It differs from true ankle clonus in its manner of commencement, in its occurrence only in an extended position of the ankle, and in its irregularity. This pseudo-clonus can be produced by any person who in the sitting position places his feet on tip-toe. The plantar reflex is difficult to obtain and often absent; when present it is always of the flexor type. The sphincters are unaffected. The paralysis may continue for years, even as long as fifteen years, and recovery ultimately follow, but adhesions in the joints and tendon sheaths are apt to form from the long disuse. Similarly there may be hysterical hemiplegia or monoplegia, and these forms are usually accompanied by contracture and anæsthesia of the paralysed limb. A very common form of hysterical paralysis is that of the adductors of the vocal cords causing hysterical aphonia. Similar functional adductor paralysis is also often seen along with genuine laryngitis. All these forms may pass off almost suddenly under the influence of some emotion or other strong stimulus, or under the influence of suggestion. Functional aphonia is often cured by the introduction of the mirror preliminary to obtaining a view of the larynx, the patient being under the belief that some therapeutic measure is being adopted. The so-called hysterical ptosis consists of spasm of the orbicularis oculi; generally bilateral, it can be at once distinguished from true ptosis by the fact that the patient resists when an attempt is made to raise the lids. In rare cases dissociated movements of the eye may occur, but diplopia never.

Spasmodic phenomena or contractures.—These may affect almost any part of the body. Most commonly one limb or segment of a

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limb is affected. The hands and feet may be so firmly contracted that it is almost impossible to undo the contracture by the strongest passive movements. Owing to the superior strength of the flexors of most joints the position is one of flexion. The position of the limbs affected with contracture differs entirely from that of the contracture of organic disease. In the upper extremity, the fingers are flexed at all joints, wrists flexed, forearm pronated, elbow flexed, and the shoulder adducted. In the lower extremity the toes are flexed, foot dropped and inverted, knee extended, and the hip extended and adducted. Flexor contracture at the hip and knee and pes cavus, which are common organic contractures, do not occur in hysterical contracture. When the spasm affects the neck muscles of one side a condition closely resembling spasmodic torticollis results. The presence of other marked signs of hysteria will serve to distinguish the two conditions. The importance of such distinction must be carefully kept in view, as surgical interference appropriate for spasmodic torticollis may greatly aggravate the hysterical form and is never to be employed in this condition. The contractures may persist during sleep. They relax under deep anæsthesia, but this is by no means peculiar to hysterical contractures. Contractures are also seen in the recti and oblique muscles of the abdominal wall, affecting parts only of the muscle. These are easily mistaken for abdominal tumours, and constitute one of the varieties of "phantom tumour." They at once disappear if an anæsthetic is given.

Tremor.—Almost every variety of tremor may be met with, from fine rhythmic tremor to coarse jerky inco-ordinate movements simulating the intention tremor of disseminated sclerosis. The tremor may constitute the chief motor symptom of a case of hysteria.

Occasionally cases are met with in which involuntary sudden movements occur repeatedly, and to these cases the term "hysterical chorea" has been applied. The movements in some cases resemble those which occur in habit spasm, while in others the aspect of the case so resembles that of true chorea that it may be impossible to decide whether a given case is hysterical or one of true chorea with hysterical manifestations. Other cases which must be included under the term hysterical chorea present movements so entirely "sui generis" as at once to suggest their hysterical nature.

Convulsive phenomena.—General convulsions are met with. They are common in France, but comparatively rare in this country. They bear a general resemblance to epileptic fits. There is a

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definite aura in many cases, which most frequently takes the form in both sexes of a pain in the flank. The aura is usually succeeded by opisthotonos, the body often resting on the occiput and the heels. This tonic stage is followed often suddenly by a struggling stage, in which the patient springs about, clutching at bystanders and often trying to bite them. It sometimes requires four or five strong men to hold her down. The movements, contrary to what is seen in the clonic stage of epileptic attacks, are purposive. Often they are extremely complicated, most appropriate histrionic attitudes being struck. Patients often talk and sing during this state, and may recite with much greater skill than they can when in their normal condition. Consciousness is, as a rule, retained during the attack, though it is generally impaired, remembrance of the attack being often absent after its occurrence. The corneal and pupillary reflexes are therefore retained, and the patient usually resists strongly the opening of the eyelids necessary for the examination of these phenomena. It must be, however, borne in mind that in rare cases consciousness may be completely lost, the pupils dilated and the corneal reflexes absent. In contrast to epileptic attacks the tongue is never bitten, the bladder is not emptied, and it is very rarely that a patient injures herself, no matter how violent her struggling may be. The chief points of distinction between hysterical and epileptic convulsions have been most clearly described by Sir William Gowers, and the following table exhibiting them is inserted here by his permission:—

	Epileptic.	Hysteroid.
Apparent cause	None	Emotion.
Warning	Any, but especially unilateral or epigastric auræ	Palpitation, malaise, choking, bilateral foot aura.
Onset	Always sudden	Often gradual.
Scream	At onset	During course.
Convulsion	Rigidity followed by "jerking," rarely rigidity alone	Rigidity or "struggling," throwing about of limbs or head, arching of back.
Biting	Tongue	Lips, hands, or other people and things.
Micturition	Frequent	Never.
Defæcation	Occasional	Never.
Talking	Never	Frequent.
Duration	A few minutes	More than ten minutes, often much longer.
Restraint necessary	To prevent accident	To control violence.
Termination	Spontaneous	Spontaneous or induced (water, etc.).

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It must be remembered that hysteroid convulsions not infrequently occur at the close of true epileptic fits and even after attacks of petit mal. In such cases there will often be a history of tongue biting or of involuntary micturition.

Retention of urine.—This is not uncommon, and is sometimes semi-voluntary, sometimes an exaggeration of the difficulty occasionally experienced by most people in relaxing the sphincter. A catheter should not be used unless absolutely necessary, and then always by the nurse. After hysteroid attacks it is usual for a large quantity of pale urine at low specific gravity to be passed. Other hysterical symptoms, most of which are referred to under the account of the diseases of the organs involved, are *hiccough*, *vomiting*, noisy *eructations*, etc.

Hysterical vomiting deserves special mention, as in some severe cases of hysteria its regular occurrence may produce a condition of extreme marasmus, and death may occur. The habit of vomiting once set up in hysterical patients is most difficult to arrest or influence by treatment.

Temperature.—It is very doubtful whether hysterical pyrexia ever occurs, most of the instances in which high temperatures have been indicated being due to the patient having tampered with the thermometer. Even severe hysteroid attacks cause no appreciable elevation of the temperature. On the other hand, in severe cases with much wasting and inanition the temperature may be abnormally low for long periods.

Diagnosis.—This is often a matter of the greatest difficulty. It must never be forgotten that patients suffering from organic diseases may readily become hysterical, and the "functional" symptoms may mask or may even precede by a long period those due to organic disease. This is especially true of disseminated sclerosis, spinal caries, and cerebral tumour. The presence of optic neuritis or atrophy is proof positive of the existence of organic disease, and nystagmus, hemianopsia, sustained ankle clonus, an extensor type of plantar reflex, and incontinence of urine are rarely, if ever, seen in cases of pure hysteria. The characteristic symptoms of hysteria will be easily recognised as such, but a confident diagnosis that the symptoms are due to hysteria and nothing else can only be given after careful watching and repeated observation. Many instances could be quoted in which hasty diagnosis of hysteria, pure and simple, led to rather harsh treatment of young women who were suffering from diseases such as cerebral tumour, which ultimately proved fatal.

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The following table shows some of the more important symptoms which serve to distinguish hysteria from organic disease. The type of the latter taken is disseminated sclerosis, which most frequently simulates hysteria. It must be most strongly insisted that, while the presence of symptoms enumerated in the third column excludes uncomplicated hysteria, their absence does not exclude organic disease, and further, that the symptoms in the second column may one and all exist with organic disease.

	Hysteria.	Disseminated Sclerosis.
Convulsions	Hysteroid (*q.v.*, p. 327)	Epileptiform (rare).
Optic discs	Normal	Atrophy (common).
Vision	Crossed amblyopia	Diminution of fields and of acuity.
Inequality of pupils Strabismus diplopia	Absent	Commonly present.
Nystagmus (marked)	Never present (albinism, old strabismus, and opacity of media being excluded)	Present.
Speech	Normal	Sometimes of a type peculiar to this disease, syllabic, etc.
Paralysis	Flaccid or spastic, hemiplegic or paraplegic	Almost always spastic and paraplegic.
Intention tremor	Never present	Common.
Foot clonus	Spurious (*q.v.*)	True.
Plantar reflex	Flexor response or absent	Extensor response.
Incontinence, etc.,[1] of urine	Never present	Almost always present.
Anæsthesia	Often present and in severe degree	Only present in advanced cases.
Girdle sensation	Never present	Common.
Pain in lower part of spine	Rare	Common.

Treatment.—The treatment of this multiform disease requires to be adapted to each particular case, and only its principles can be here given. The most important element is the securing of the complete confidence of the patient and her complete obedience. Unfortunately their temperament and their relish for the privileges of an invalid do not predispose them to assist their adviser in this way. The patient's surroundings should be investigated, and any cause of emotional disturbance should, if possible, be removed, or the patient may be sent away from them

[1] The term incontinence, etc., is used here collectively for precipitate micturition, delayed micturition, retention, unconscious micturition, and involuntary micturition.

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for a time in charge of some firm but kind nurse or other person to whose authority the patient will yield. Relatives do not make good nurses for hysterical patients. Both the medical man and the nurse must remember that hysteria is a real complaint and not mere naughtiness, although as a rule complicated by it, and consistent kind firmness rather than alternate petting and severity must be employed. The general health must be kept up and plenty of food taken.

Where motor symptoms are present electricity in the form of faradism or static electricity and massage are most useful. In the application of such treatment it is most important that the patient should expect the desired beneficial result to occur. The applier of treatment should strive that some improvement, however, should occur at each application and should demonstrate it for the encouragement of the patient.

Suggestion is important in the treatment of hysteria, not only in the removal of symptoms, but also in their production, and the suggestion of symptoms during the physical examination of the patient must be carefully avoided. We have seen total blindness, superficial anæsthesia, aphonia, and monoplegias produced by the involuntary suggestion which may result during physical examination.

Hypnotism is said to have been very useful in some cases. In cases under the care of one of us it certainly on occasions removed symptoms, but without doubt tended to increase the mental instability, which is a fundamental factor in this disease. In several cases severe mental derangement followed its use.

Metallo-therapy has had its ardent supporters, and is perhaps as useful as any other form of faith-healing.

Drugs are required chiefly to promote the appetite or as sedatives. The old custom of giving all manner of disgusting mixtures as semi-punitive measures is a mediæval survival which should be abandoned. Valerian and asafœtida appear to have considerable value, and their nauseous taste can be obviated by giving the asafœtida in pill, and valerian may be given in pill in the form of valerianate of zinc in two or three grain doses. For obstinate cases no plan of treatment is better than the "Weir-Mitchell treatment." This consists of four distinct parts, which *must* be employed together if success is desired—(*a*) Rest in bed. (*b*) Complete isolation from relatives, and at any rate at first even correspondence may be prohibited. A cheerful and tactful nurse is essential. (*c*) Massage. (*d*) Over-feeding with eggs and milk in addition to ordinary food. The duration of a course of

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treatment is from a month to six weeks, the restrictions as to isolation being gradually relaxed towards the end of the time.

NEURASTHENIA

SYN. NERVOUS EXHAUSTION, NERVOUS BREAKDOWN, SPINAL WEAKNESS, ETC.

This may be described as a mental condition associated with alteration of the bodily nutrition which is expressed chiefly by disability for mental work, easily induced fatigue, and a train of varied sensory symptoms for which no physical basis can be discovered, but which absorb the attention of the patient out of all proportion to their severity. Although the symptoms are very variable, there is sufficient agreement between the different cases to justify their being grouped together.

Etiology.—The disease is most prevalent among busy civilised communities, although by no means confined to them, and appears to be more frequent in the United States than in Europe. The most common *age* is between twenty and forty. Males are affected three times as often as females. Although there is often a nervous family history, *heredity* plays a much less prominent part than in other neuroses or mental affections. But occasionally many members of the same family suffer and direct heredity is obvious. In some of these families there has been consanguinity of parents and it seems that this is a possible factor in the disease. By far the most frequent cause is *prolonged stress* due to grief, worry, overwork, or to an association of these three factors. Sudden *shock*, caused by bad news, or the sight of some catastrophe, is a not uncommon cause. *Railway and carriage accidents*, in which these factors are accompanied by a physical jar, are followed by neurasthenia of so definite a type that it is known by the special name of "traumatic neurasthenia" or "railway spine." The morphine and cocaine habits may of themselves produce the most profound conditions of neurasthenia, and when adopted by the neurasthenic, as happens not infrequently, greatly aggravate his symptoms. Since these drugs cause temporary relief of symptoms and subsequent and lasting physical prostration, the most vicious of all cycles is prone to be set up. *Alcoholism*, although likely to lead indirectly to such stress as would induce the disease, seems

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to have little direct influence. The same may be said of *sexual excess* and masturbation, although the lying pamphlets of quacks are responsible for a certain number of cases through the terror and anxiety they induce. But in some cases such excess seems to be a symptom of the neurasthenia, as the combined result of self-absorption and the physical condition of irritable weakness. Lastly, some *febrile diseases* are apt to cause neurasthenia. Of these *influenza* stands pre-eminent, and enteric fever is also a frequent antecedent. It is most frequent after severe attacks, but may occur after quite mild forms.

The symptoms of neurasthenia often arise in the course of organic disease, sometimes grave, sometimes trifling and not infrequently of such a nature as to escape detection. Among such conditions early phthisis, Addison's disease, granular kidney, Graves' disease, dilatation of the stomach, floating kidney, and enteroptosis may be mentioned. Of especial importance in this connection is osteoarthritis, for many of the subjects of this disease present typical symptoms of neurasthenia. Further, many intractable cases of neurasthenia present the clammy sweating skin, the peripheral trophic changes, the anæmia, the yellow-gray-white complexion, the curious mottling of the skin (Spender's spots), the tachycardia and the slight pyrexia of osteoarthritis, but the joint changes may be either entirely absent or detectable only upon the most careful examination, or a case may present any degree of polyarthritis up to the most severe type. Whatever the connection between osteoarthritis and neurasthenia may be it is interesting, since in both diseases a toxic agent has been suggested as the cause, and with considerable weight.

Symptomatology.—The *physical condition* varies. The patient may be full fleshed, but more often is sparely built, restless, and fidgety; and in some there is very marked wasting. The appetite is capricious, and there is usually constipation, alternating with relaxation of the bowels. Broken or unrefreshing sleep adds to the patient's misery.

General mental condition.—Speaking generally, the patients are depressed, often emotional, with an incontrollable tendency to discuss their symptoms in detail, dwelling unnecessarily on descriptions of their secretions or evacuations. There is nearly always a curious tendency to disagree with any suggestion made as to treatment, and an air of triumphant resignation when they assure their medical adviser at their next visit that they are worse. Their self-absorption leads them to choose for light reading any literature bearing on

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diseases with symptoms akin to theirs, and their extensive but confused acquaintance with medical terms is as characteristic as it is trying.

Psychical symptoms.—There is nearly always incapacity for work requiring mental effort. This is due to several factors. Not only does the patient find that with all his effort the work is ill done, but a sense of fatigue attacks him before he has fairly begun. His memory is unreliable, and there is such indecision that he is unable to arrange his own work or that of others. He finds it impossible to concentrate his mind on the matter in hand, and even in reading the newspaper he finds that he has got to the bottom of a column without having grasped its meaning. These defects interfere with the patient's occupation, worry is superadded and aggravates the mental condition, and a vicious cycle is set up. Constant restlessness is a marked feature in some cases.

Claustrophobia and agoraphobia.—In connection with the mental indecision referred to above, there are two special manifestations which have received special names. Claustrophobia is the condition in which the patient has a dread of being in an enclosed space, such as a narrow street between tall houses. The opposite condition is seen in agoraphobia, in which the patient is utterly unable, from a terror which he cannot explain to himself, to cross open spaces, such as market-places or fields. The symptoms are met with in other mental conditions.

The sensory processes generally are exaggerated. Loud voices and bright scenes are usually annoying, and there is increased appreciation of all visceral sensations, which appear magnified and distorted.

In addition to this mental condition there is a complex tangle of symptoms which baffles any attempt at clear description. The individual threads may be unravelled and examined separately, but it must be remembered that in practice the appearance of the tangled skein varies indefinitely. The symptoms, a few or many of which may be present, are most conveniently divided into (1) cephalic, (2) special sense symptoms, (3) spinal, (4) vascular, (5) muscular, (6) visceral, (7) sexual.

1. *Cephalic sensations.*—Headache, usually most severe on the vertex or occiput. Often there is a sense of fulness or of external pressure, or there may be a sense of fulness, suggesting to the patient that there is a tumour beneath the skull. The headache is usually increased by stooping, and also by mental effort. It seldom interferes with sleep, and is usually least troublesome after

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meals. Not infrequently it is accompanied by cutaneous tenderness of the scalp.

2. *Special sense symptoms*—(*a*) Hearing.—Deafness is rare. During the paroxysms of headache swimming sensations in the head and buzzing of the ears are often complained of, and tinnitus is a frequent symptom.

(*b*) Vision.—Failure of accommodation (asthenopia), in which there is indistinctness of vision ("letters running into one another") without error of refraction, is common. Muscæ volitantes are frequently the cause of much worry to the neurasthenic. Affections of the pupil, diplopia, and nystagmus, while frequent in the early stage of organic disease, are not symptoms of neurasthenia. The visual field is often contracted, especially in traumatic cases.

(*c*) Common Sensation.—Cutaneous hyperæsthesia, especially over the spine, is extremely common. Its site varies from time to time, and if the patient's attention is diverted he can usually bear firm pressure without wincing. Tingling, burning, or itching sensations in various parts are often troublesome.

3. *Spinal symptoms.*—The spinal symptoms are often very prominent, and consequently patients are often said to be suffering from "spinal weakness," "irritable spine," and so forth. Aching pains up and down the spine, or across the loins, are complained of by most, and they are often accompanied by superficial cutaneous tenderness, as described above. Jars and shaking aggravate the pain, and hence spinal caries is often suspected. The knee-jerks, elbow-jerks, and wrist-jerks are nearly always greatly exaggerated, but true ankle clonus is rarely, if ever, obtained. The superficial reflexes are difficult to elicit. An extensor form of the plantar reflex (Babinski's sign) is never obtained. The deep reflexes are unaffected, except that sometimes there may be retention of urine, as in hysteria. Incontinence is always indicative of organic disease.

4. *Vascular symptoms.*—Symptoms referred to the heart and circulation are very common and alarming to the patient, and are usually aggravated by accompanying dyspepsia. Sometimes they are due to the patient smoking more tobacco than usual in order to "quiet his nerves." Præcordial pain, or uneasiness, inframammary stabbing pain, palpitation, or attacks of pain with anxiety simulating angina are the most frequent symptoms. The pulse in neurasthenics is usually rather rapid and of low tension. The patient is often annoyed by sudden flushing of various parts, especially of the face and trunk, and coldness of the hands and feet are often troublesome symptoms. Abdominal throbbing is

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sometimes complained of, and in these cases a markedly pulsating aorta is commonly present.

5. *Muscular symptoms.*—There is usually much flabbiness of the muscles from loss of tone, and there may or may not be wasting. Tremor of the hands when the arms are extended is common; it is fine in character and resembles that seen in exophthalmic goître. Occasionally twitching of the muscles may be seen, occurring at irregular intervals sufficient to tighten up the tendons but not to move the limb.

Arthritis.—There is a statement, somewhat general in recent medical literature in this country, to the effect that joint changes are met with not rarely in hysteria, but that in neurasthenia such arthritic lesions are not met with. An investigation carried out by one of us upon a large number of cases of hysteria and neurasthenia at the National Hospital for Paralysis, to the Medical Staff of which institution we are indebted for permission to record the observations, showed that changes in the joints were comparatively common in cases of neurasthenia, while in hysteria they were very rare and amounted for the most part to rest and contracture adhesions, in cases of long-standing paralysis and inanition.

6. *Visceral troubles and sensations.*—Dyspeptic symptoms are nearly always present, so that some regard them as primary. There is much flatulence and a "sinking" epigastric sensation, which is relieved by taking food. Constipation, alternating with some relaxation, adds to the patient's discomfort and anxiety. There is often frequency of micturition, the urine being pale, of a low specific gravity (1010 to 1012), and containing excess of phosphates.

7. *Sexual symptoms* are sometimes complained of by men. Most often they are alarmed by nocturnal emissions which may be accompanied by incompetence for the normal act. Such patients are depressed, their minds having been poisoned by the pamphlets of quacks. They often insist that they have some loss of semen at the end of micturition, but microscopic examination of the urine rarely bears this out.

TRAUMATIC NEURASTHENIA ("Railway Spine," "Concussion of Spine").—Neurasthenia frequently occurs as a sequel of such accidents as railway collisions, carriage upsets, and accidents in the hunting field, in which there is a physical shaking as well as mental shock.

The symptoms in such cases are those described in the preceding section, but the picture presented is somewhat more

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uniform. One of the most important features is the interval which commonly separates the accident from the onset of nervous symptoms. The victim of a railway smash may assist his fellow passengers and may continue his journey home without any complaint; the huntsman may remount and rejoin the hunt: definite symptoms manifesting themselves only after a latent period varying in duration from a day to several weeks. When the disorder is well developed the psychical symptoms (p. 333) are most pronounced and are accompanied by loss of flesh and much physical weakness. Mental and bodily fatigue are easily induced. Self-distrust, hesitancy and indecision, and loss of memory are usually prominent symptoms, and there is insomnia or broken, unrefreshing sleep. Uncomfortable head sensations (pressure, buzzing, vertical headache, etc.) and spinal pain and tenderness are complained of. The knee and elbow-jerks are much exaggerated, but true ankle clonus is probably never present unless there is some organic trouble. In addition to these, which are more or less constant, any of the symptoms described as occurring in ordinary neurasthenia may be present.

HYPOCHONDRIASIS.—Sometimes the visceral sensations are so pronounced that it is regarded as a separate disease—hypochondriasis. The patient's whole time is taken up in consideration of the various organs which he firmly believes to be diseased. It is useless for his medical attendant to assure him that the organ is sound. If he has palpitation, he demeans himself as becomes a cardiac invalid; if he has a cough, he adds to it a loud barking sound, wraps himself up, and insists on it that he is "in a decline," and so on. He has in fact delusions about his health, although, as his intellect is not otherwise disordered, it is not customary to speak of them as insane. The following transcript of a hypochondriac's own description of her symptoms is characteristic: "I have been a sufferer for years; in one day I have about fifty diseases, my head and stomach especially. I cannot eat. If I eat the lightest food then I have indigestion, so I don't eat at all. I can't sleep, and if I do sleep for a few minutes I have such horrible dreams. I dream I am here, there, and everywhere. If I sleep a little I am always a-moaning for pains all over my body. First my back, I can't stoop, then my chest, all down my sides, my shoulders, my legs: then the pain goes towards my stomach and head, and the pain stops there. I have been to doctors, and they told me not to eat meat, so I have not eaten meat for years.

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I see there is no improvement, but the reverse. I can't even wash a cup; I can't stand on my feet through the pain that I have. The principal pain is in my head and stomach. I always thought that I had a complaint in my heart, but the doctors have examined me and said my heart was well. . . . Even when I wash myself the pain I have is horrible; sometimes I feel so heavy that I can't move myself, and I also have a burning in my body. This burning kills me altogether, for it leaves me very weak. I have such pains that even the nails of my fingers ache." The suffering which such patients endure is very great and accompanied by so much mental depression that life is a burden to them. They not infrequently ultimately become insane. Treatment is conducted on the same lines as that for neurasthenia, but the prognosis is not favourable when the symptoms are pronounced.

Pathology.—No morbid anatomical changes are to be found in the central nervous system. The disease appears to be due to some functional disturbance of the higher cerebral centres. Some hold that it is due to an "auto-intoxication" from absorption of the products of a faulty digestion. But although dyspepsia is a frequent accompaniment of neurasthenia and the two react on one another, it is the nervous condition which is usually primary.

Diagnosis.—As in the case of hysteria, it must always be borne in mind that neurasthenia and grave organic disease may co-exist, and in every case most careful search must be made for any sign of nervous or visceral organic disease.

From hysteria on the one hand and hypochondriasis on the other it is impossible to draw any definite boundary line. It is rare to meet with definite anæsthesia, with convulsions, or with mimetic palsies in neurasthenia, while they are common in hysteria. From hypochondriasis or melancholia the distinction is even slighter, as all neurasthenics at times are hypochondriacal and depressed.

The train of symptoms described earlier, when well developed, is sufficiently characteristic to make the diagnosis easy. But the disease is often of slow onset, and its early stages may easily be confused with those of other insidious disorders in which gradual loss of strength and energy may precede the characteristic symptoms. Such are cerebral tumour, disseminated sclerosis, diabetes, chronic Bright's disease, exophthalmic goître, phthisis, and malignant disease of the abdominal viscera. Examination of the fundus oculi, of the abdomen, and of the urine, should never be omitted. On

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the other hand, the visceral symptoms may be so prominent that the medical man may be led wrongly to suspect organic disease. This is the more likely since these patients have often read medical descriptions of such diseases, and from "suggestion" begin to experience the symptoms of heart disease, angina pectoris, or cancer of the stomach. A careful examination may show that the suspected organs are sound, and conviction of this on the part of the patient will usually be followed by rapid improvement of the symptoms.

Prognosis.—If the attack has come on in a previously healthy individual from some definite cause, such as worry, *and the cause can be removed*, spontaneous recovery may occur. Unfortunately these conditions rarely obtain. The patients usually have a restless, neurotic temperament, and often a neurotic inheritance; the conditions producing worry or other stress may not be of a temporary character; and the illness, by interfering with his income or prospects, may increase his worries. The prognosis, as a rule, is best when the patient's means and circumstances allow of his changing his whole surroundings for a time by indulging in travel and change of scene, or a course of mental and physical rest, as in the Weir-Mitchell treatment. The duration of the disease is very variable, but the patient is fortunate if its duration can be measured in weeks, and it may run on for years. It does not tend to shorten life, but may make it a burden to the victim and his associates. In not a few cases it leads ultimately to definite mental derangement. In traumatic cases there is often rapid improvement after the strain of a law-suit is over, whichever way the verdict goes. But this is by no means always the case, even where the patient has succeeded in recovering ample damages.

Treatment.—The principles of treatment are the same as those for hysteria. The first thing is to gain the confidence of the patient by a thorough investigation of his symptoms. The cause of his mental condition should be inquired for, and if possible removed. Rest, change, and distraction should be ordered in different cases, but routine prescriptions of foreign travel, sea voyages, etc., should be avoided, as each individual requires adaptation of treatment to his special needs and circumstances. The man who has been over-worked at business, and yet is carrying on religious or philanthropic work in the evening, may be advised to substitute physical exercise for the latter for a time, and a neurasthenic whose time is not filled up may have his interests and activity directed into philanthropic, literary, or scientific pursuits

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to prevent morbid attention to his own symptoms. But in many cases there is incapacity for both work and recreation—"his nerve is gone." In such cases a course of "Weir-Mitchell treatment," carried out in its entirety, will usually give most satisfactory results. This is especially true of the traumatic cases, and it succeeds not only when the condition of bodily nourishment is below par but also in fat neurasthenics whose weight is often reduced by the massage. Hydrotherapeutics, balneotherapeutics, and the employment of graduated exercises give excellent results in some cases.

In the way of drugs, tonics, especially iron and strychnine, are most useful. The latter drug must, however, be used with care, and in small doses gradually increased, as it is liable to increase the irritability, restlessness, and sleeplessness of the patients. They are conveniently given in the form of syrup of the phosphates or hypophosphites. By far the most useful sedative drugs are the bromides, which may conveniently be given in combination with strychnine. Again, neurasthenia is very frequently accompanied by vasomotor irritability and disturbances, but it is probable that these are only secondary conditions and that the primary pathogeny of the condition is not in the vascular system. Opium, morphine, and cocaine are to be avoided, as drug habit is easily set up.

CHOREA

Syn. Sydenham's Chorea, St. Vitus's Dance

A disease chiefly met with among children, characterised by involuntary, irregular, inco-ordinate movements in one or more limbs or of the whole body, and by a tendency to endocarditis, and other rheumatic manifestations. Chorea hardly ever occurs in children under three years of age.

Etiology.—It chiefly attacks those between the *age* of five and ten years, although second attacks are common after the latter age. Girls are attacked three times as frequently as boys, and after the second decade chorea is almost confined to females.

A *neuropathic heredity* is important, hysteria, epilepsy, and insanity being most common, and it is in families where neuropathic and rheumatic heredity are met with together that the incidence of chorea seems to be greatest.

Race has an important influence, for chorea is rarely found

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among negroes, and according to Osler is never met with among pure bred North American Indians, though it occurs among the half breeds. On the other hand, it is very common in Hebrew races.

There can be no doubt as to the close association of chorea and *rheumatism.* Not only is there a family history of rheumatism in the majority of the cases, but a personal history of previous "growing pains," if not of actual joint swelling, can often be elicited, and in others, if the patients are followed up, many more of them develop rheumatism. Thus Dr. Batten found that out of 115 cases of chorea treated at the Great Ormond Street Children's Hospital there was a history of previous rheumatism in 32 per cent. He followed these cases up three years later, and found that in that short time many more had suffered from rheumatism, bringing the percentage up to 43.5. It must also be remembered that not only do choreic patients suffer from endo- and pericarditis of the rheumatic type, but they may also have subcutaneous nodules and erythematous rashes exactly like those occurring during the course of acute rheumatism. In fact, chorea may be looked upon as a nervous manifestation of rheumatism. (See Vol. I. art. Rheumatism.)

It has been stated (Ross) that chorea frequently occurs after scarlet fever, but as the result of detailed investigation it appears that the relation of the chorea is with rheumatism, itself an associate of scarlet fever, and that chorea is not especially incident upon those who have suffered with scarlet fever and have at no time presented symptoms of rheumatism.

Pregnancy is occasionally a predisposing cause, owing no doubt to the mental strain associated with it; and hence it is most common with first pregnancies, particularly when the mothers are unmarried. In some of the pregnant cases the chorea is of a violent or maniacal type, endangering life; in others it runs an ordinary course. It occurs most frequently during the first three months of pregnancy.

Exciting causes.—In many cases no exciting causes can be traced, but where they are present they are usually of an emotional character. Patients often complain of having been suddenly terrified by animals, by some practical joke, or by witnessing an accident. In some cases the movements follow at once upon some emotional disturbance, and this sudden onset is to be explained by the depressing influence of the emotion upon a nervous system, which as a result of the rheumatic poison, for an instance, is in a condition of

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high instability. But in many cases in which it is supposed that some fright brought on chorea it will be found on careful inquiry that there had been previous slight movements and that the fright suddenly aggravated the condition. Punishments at school or at home are often given as causes, but very often the punishment is, so to speak, the result, and not the cause of the chorea. It has been said that every child with chorea may look forward to three whippings, one from her teacher for not sitting still, another from her mother for dropping the tea-cups, and a third from her grandmother for making faces at her. In older children the strain of nursing an invalid mother has in several cases provoked an attack. Imitation was formerly regarded as a frequent cause of chorea, but it is doubtful whether it is ever contracted in this way. No cases have arisen in this way at the Great Ormond Street Hospital during thirty years.

School life.—The influence of over-pressure in schools has been insisted on by many writers, but in the writer's opinion this factor has been exaggerated. Chorea is sometimes started and always intensified by emotion, and anxiety lest she should fail in an examination, her resentment at the injustice of punishment for the fidgetiness of the early stage of chorea, or her chagrin at punishment justly incurred, may cause or aggravate chorea at school as similar emotions would in other circumstances. In a small number of cases, however, the stress imposed by the child's lessons being beyond her capacity has seemed directly responsible for the break-down. In any case, after the early signs of chorea have shown themselves, school is the worst place for the child, and a return to school before she has completely recovered from the disease is nearly certain to bring about a relapse.

Morbid anatomy.—The seat of the disturbance in chorea—the cerebral hemispheres—is indicated by the quasi-volitional nature of the movements, their frequent hemiplegic commencement and distribution, and the frequently associated hemiparesis. The excitation of an attack by emotional disturbances, the mental state during an attack, and the occasional association of acute mania also point to involvement of the highest parts of the nervous system.

In some of the early post-mortem investigations of the disease small foci of softening in the region of the basal ganglia and corpora striata were found. From this it was argued that these structures were the seat of multiple embolisms from associated

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endocarditis, and thus arose the "embolic theory" of chorea. This theory was further supported by the production of spontaneous irregular movements in animals by the introduction of starch grains into the circulation. Further investigation has entirely disproved this theory. Lesions in the region of the corpora striata are rare in chorea and are often its consequence, not its cause. Further, a cause for embolism is often absent.

Quite recently Drs. Poynton and Paine have isolated a diplococcus from the joints, pericardial cavity, and heart muscles of cases of acute rheumatism. This organism, when injected into rabbits, produced acute arthritis, acute endocarditis and in some cases chorea. From the affected tissues of these animals, again, the diplococci were obtained in pure culture. Sections of the cerebral cortex of the choreic animals, and also of a patient who died during acute chorea, showed masses of these diplococci in connection with the small arterioles. The investigations of Pianese, Wasserman, the late Dr. Charlewood Turner, and Dr. B. Abrahams also tend in the same direction.

Further, the dependence of the movements characteristic of chorea upon multiple small lesions in the cortex is also suggested by the fact that the similar movements in Huntington's chorea, and the so-called choreic movements of diplegia are, in the present state of our knowledge, to be attributed to scattered cortical lesions of small size.

Symptoms.—*Motor*—(*a*) Spontaneous, arhythmic, involuntary, complex movements; (*b*) Inco-ordination; (*c*) Muscular weakness. These may exist together, but they usually come on in the order given. The spontaneous movements at first are best described as "exaggerated fidgetiness" and are rarely recognised by parents or teachers. They usually commence in the face and hands, rarely in the legs, and in many cases one side of the body is affected long before the other, and the unilateral character of the movements may persist throughout the attack, the condition known as *hemichorea*. The movements are worse when the child is excited, cease during sleep, and at first can be inhibited by a strong effort, as when the child tries to write. After writing a few words, however, the movements return. The movements are usually sudden, and always complex. They are irregular in nature, in seat, in rhythm, and in range, and are purposive in character, *i.e.* they resemble the common voluntary movements and so contrast strongly with tremor, intention movements and convulsive movements, which are not purposive, and with simple inco-ordination, where the irregular

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movement is not spontaneous. The smooth regularity of the normal respiratory movements is broken at one or more places in each respiratory phase, and frequently the respirations are irregularly spaced. A peculiar squirming movement of the trunk superadded to this produced a picture difficult to describe but highly characteristic of chorea. Thus, in the face there are grimaces which are constantly varying, in contrast to the simple repeated blinking, etc., seen in habit spasm (p. 348). In severe cases all the voluntary muscles may be affected, the head, arms, legs and trunk being jerked about, so that the patient has to be restrained to prevent her falling out of bed. She is quite unable to feed herself, and, owing to the movements of her face and tongue, it is very difficult to feed her, and the inco-ordination of the muscles of deglutition makes it difficult for her to swallow, particularly in the case of fluids. The movements may be so violent as to entirely prevent sleep for long periods. Inco-ordination is usually present along with the spontaneous movements, but it may occasionally precede them, there being slowness in dressing, awkwardness in writing or unsteady gait before fidgetiness is observed. Inco-ordination is best seen in the gait, in handwriting, and in the movements of respiration, the movements of the diaphragm being irregular and no longer synchronous with those of the intercostals, often alternating with them. Muscular weakness is seldom conspicuous until the choreic movements have persisted for some time. It is, however, in rare instances an early symptom, and may obscure the diagnosis. It is most commonly hemiplegic, but may affect one or all of the limbs (*chorea mollis* or *chorea paralytica*).

The weakness in choreic hemiplegia is usually upon the left side, but in left-handed people it is the right side which suffers most. Marked general asthenia is usually present, and choreic movements may be entirely absent so far as face and limbs are concerned, but the above-described peculiarity of respiratory movements is generally present. The absence of choreic movements is a frequent cause of error in diagnosis and it is important to bear in mind the statement of Sir William Gowers that a hemiplegia of subacute onset (three to seven days) occurring before puberty is most often chorea. Choreic hemiplegia may attack first one side and afterwards the other.

Speech is usually affected in severe cases. There may be indistinctness of articulation owing to the jerking of the labial and lingual muscles, or there may be inability even to phonate owing to inco-ordination of the laryngeal muscles. In rare cases

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there appears to be interference with the functions of the motor speech centre and true aphasia, or even complete aphemia. In consequence of jerking of the ocular muscles *nystagmus* may be seen in severe cases.

Sensory changes are unimportant. Slight blunting of sensation can occasionally be demonstrated, especially in cases of hemichorea, and occasionally there are tinglings of the extremities. Ordinary rheumatic pains are frequently met with.

Reflexes.—The pupils are dilated, but react to light. There is frequently difficulty in swallowing. The other organic reflexes are normal. The knee-jerks are usually active, and often there is a "sustained" knee-jerk, the leg being held extended for about a second after the patella tendon has been struck. Occasionally the knee-jerk is lost for a time.

Mental condition.—Chorea is most common among bright, intelligent girls. During the attack they become emotional and often irritable. Delirium is rare. After severe attacks there is often some temporary mental enfeeblement. Chorea in adults is sometimes followed by acute mania.

Cardiac murmurs are heard in the majority of cases, and occur both with and without a history of previous joint pains. In some they are hæmic and attributable to the anæmia which is usually present, being heard chiefly at the base, and disappearing with the anæmia. But the majority are due to endocarditis affecting the mitral valve, or occasionally the aortic. This endocarditis differs in no way from that due to rheumatism alone, and in children, owing to the readiness with which compensation occurs, usually gives rise to no symptoms, and may be overlooked unless the heart be carefully examined. More or less pericarditis has also been present in the majority of fatal cases. It is less frequent than endocarditis, and is rarely severe. Irregularity of the cardiac rhythm, due to disordered nervous control, is frequently observed, and occurs even where there is no endocarditis.

The *temperature* is usually normal, unless there is some inflammation of the heart or pericardium, or some recrudescence of articular rheumatism. There is also pyrexia in the cases accompanied by mental excitement.

Even in mild attacks the symptoms rarely subside completely in less than six weeks, and may last three or six months. Relapses, due usually to some emotional cause, are common. Second and third attacks are not rare, and usually occur at about the same time of year as the first attack.

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Diagnosis.—When the disease is well marked there is little difficulty in diagnosis. When the movements are slight they may usually be brought out by making the child stand with the arms raised above the head, and the tongue protruded. The variability and complexity of the movements distinguish it from habit spasm (p. 348), with which it is sometimes confused. Weak-minded children often have some irregular, inco-ordinated movements, but these have existed all their lives, while chorea comes on as definite attacks. The most difficult cases are those in which there is much paralysis, but careful inquiry as to the mode of onset should prevent error.

Prognosis.—The prognosis as regards the chorea is good in the great majority of cases, although any cardiac disability left by the endocarditis may be permanent. The prognosis is less favourable when the disease occurs after puberty. Exhaustion from want of sleep, or from taking insufficient food, is the most frequent cause of death, or it may occur from cardiac complications.

Treatment.—In the treatment of chorea, rest and good feeding are by far the most important factors. The children should be kept resting on the couch, or in severe cases entirely in bed. They should not be tied or restrained, as this increases the violence of the movements. Bolsters should be placed to prevent them from bruising themselves by knocking their limbs against the edge of the bed, and if there is any risk of their jerking themselves out of bed they should be placed on a mattress on the floor. The child should wear woollen garments of such a character that he is not exposed to the cold air if he throws the bed-clothes off. Quiet should be preserved in the room, and many visits discouraged. If the movements are severe a water-bed is desirable, as the constant friction is apt to cause abrasions of the skin, which readily become bed-sores. Abundant food must be given. Fluids are often swallowed with difficulty, but pultaceous food is more readily taken. Porridge, bread and milk, sponge cake and milk, mashed potato with pounded fish or minced meat are convenient modes of giving food. If there is much exhaustion alcoholic stimulants should be given. The skin should be sponged over several times a day, especially before the child tries to sleep. Gentle stroking of the whole body after sponging has a soothing effect and assists sleep. In chronic cases cold douching of the spine often hastens recovery, but care must be taken not to frighten the child, who should stand in a foot-bath of hot water during the application.

It must be confessed that the drug treatment of chorea is

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unsatisfactory. The drug which is most in favour is arsenic, given in doses of from three to five minims three times a day, but it cannot be pretended that it has any specific influence comparable to that of the bromides in epilepsy or salicylates in acute rheumatism. It has been proposed to give it in increasing doses till definite toxic effects showed themselves, such as vomiting. But experience has shown that this heroic dosing is not without risk, several cases having developed arsenical neuritis, and in others a brown pigmentation of the skin, like that met with in cases of Addison's disease, has followed. Other tonics, such as nux vomica and quinine, are often given, and a very large number of drugs have had a temporary vogue, and been discarded. Most reliance has to be placed on the general treatment described above. Sleeplessness may require the use of hypnotics. A combination of chloralamide or chloral with potassium bromide will usually be found most effectual. If symptoms of articular rheumatism manifest themselves salicylates must be given. Prolonged and very severe paroxysms of spasm may be relieved by the maintenance, even for some hours, of chloroform anæsthesia.

Chronic Chorea

A term loosely employed to describe forms of chorea coming on in adults after middle life, and which have a chronic and progressive course. The cases fall into two distinct categories, senile chorea and Huntington's chorea. They have no relation with Sydenham's chorea, which is a disease of early life.

Senile Chorea

A rare condition characterised by general choreic movements coming on after middle life, and more commonly in the aged. It has no relation to rheumatism. The cases differ from those of Huntington's chorea (*a*) in the absence of hereditary tendency; (*b*) in the absence of associated mental change; (*c*) in showing a tendency to recovery.

Huntington's Chorea—Hereditary Chorea

A rare disease, characterised by the appearance in adult life of general choreic movements associated with progressive dementia. The disease was first described in 1872 by Dr. Huntington, an American physician.

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The affection is hereditary, often affecting several children of the same parents, and being sometimes traceable backwards through four generations of ancestors. It affects both sexes equally.

The symptoms make their appearance in the fourth decade of life, and consist in slight general choreic movements, precisely similar to the movements of Sydenham's chorea but somewhat slower. With the appearance of these movements inco-ordination of motion generally becomes apparent. In more advanced cases all the muscles of the body become affected; speech becomes clumsy and slurred; and the gait, unsteady and lurching, resembles somewhat that of a drunken person. The movements, which are present during rest, are, in the early stages, decreased, and in the later stages usually increased by exertion. Accompanying these symptoms is a gradually progressive dementia, often with suicidal tendencies. Sometimes attacks of acute mania occur and occasionally the mental symptoms precede the motor. In the latest stages spasticity of the limbs with increase of the deep reflexes occurs from involvement of the pyramidal system. Death may not occur till old age is reached, and is then preceded by complete dementia and general emaciation.

Morbid anatomy. — The constant condition found post-mortem is a sclerosis of the cerebral cortex in small widely-scattered patches (scattered miliary sclerosis), associated with chronic pia-arachnitis. It is probable that the sclerosis is due to some vascular condition and is not a primary atrophy of the neurones. The essential pathology of the disease is unknown.

Treatment has no effect either in arresting or removing the disease.

Congenital Chorea

This term has been applied to certain cases in which irregular, quick, involuntary movements of the limbs, dating from birth, occur. In the majority of the cases rigidity of the limbs, with increased deep reflexes, is associated, and such cases belong to the class of cerebral diplegias. In some, however, there is no rigidity, and these are regarded as being of the same category as Huntington's chorea. In neither variety do the movements in any way resemble those of true chorea.

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Chorea Major

Syn. Epidemic Chorea, Dancing Mania

This is the original disease, to which the name chorea (χορεία, a dance) was given. It occurred in epidemic form in the Middle Ages, associated with religious excitement, and is a hysterical manifestation, having no connection with the disease now known as chorea. It was also the original St. Vitus's dance, it being customary for those affected by the dancing mania to repair to a shrine of St. Vitus, who was the patron saint of actors and dancers.

HABIT SPASM

Syn. Habit Chorea, Tic Convulsif

A common affection in late childhood, characterised by the frequent repetition of some simple muscular action, the movement being always the same and not varied, as in true chorea, with which it is often confused. The most common forms are winking, snapping of the eyelids, sniffing, tossing the chin in the air. Occasionally the trick consists of a sudden movement of the tongue or larynx with an unpleasant fidgeting sound.

The attacks are nearly always in neurotic children and not infrequently have been started by some local cause (the presence of adenoid vegetations in the naso-pharynx being a not infrequent one), and afterwards have become involuntary, the subjects being unconscious of them. The blinking may be started by some conjunctivitis or error of refraction, sniffing by nasal catarrh or obstruction, and tossing the head in girls by long hair catching in the dress, etc. Some cases owe their origin to imitation, not necessarily a direct imitation, but rather an irresistible tendency to give vent to pent-up feelings of unrest engendered by the witnessing of similar movements in another. A lowered state of general health, or some influence depressing the nervous system, such as fright or mental overwork, are often present. Not uncommonly the condition develops on a child's first going to school. Optical defects are certainly responsible in some cases.

Symptoms.—The movements, which are characterised by the

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rapidity with which they are executed, are simple and regular, but more than one kind of movement may be present. They may follow one another at intervals of a few minutes usually, but may be so frequently repeated as to become almost incessant.

The face, head, and neck are most frequently affected, blinking movements being most common of all. The upper limbs are commonly affected, shrugging movements of the shoulders being the most frequent form met with. The lower extremities are rarely affected. Often the respiratory muscles and larynx are involved. The movements are increased by excitement and cease during sleep. Both in simple habit spasm and in "tic convulsif" there may be an irresistible impulse to explosive utterances; obscene words may be used (coprolalia), or certain sounds may be continually repeated (echolalia), or there may be impulses to imitate certain gestures (echokinesis).

In France many cases have occurred in which habit spasms of more complex nature than above described are accompanied by uncontrollable utterances, an impulsive tendency to imitate by acts or words, and a peculiar mental state in which imperative ideas play an important part. The condition is most frequently a hereditary one, often directly so. To this condition the term "tic convulsif" is now confined.

Treatment.—The exciting cause should be searched for and if possible removed. Great patience is required in correcting the patients, as they are unaware when they twitch. They must be induced by a judicious combination of rewards and deprivations to make a strong effort to conquer the habit. If it have existed for several years, the "trick" is apt to become permanent. The general health must be improved by tonics, change of air, etc. No drug is known to have any special influence on the condition.

Head-nodding, etc.

The term head-nodding is perhaps the most convenient one for an affection of early infancy, in which nodding lateral or rotatory movements of the head occur from time to time. Nystagmus is also frequently present. The terms *spasmus nutans*, salaam spasm and head-jerking have also been applied to this condition. The affection is congenital, but only in some instances can any heredity be traced. The causal factors are obscure. The movements make their appearance almost always between the age of four and ten

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months. Congenital syphilis and birth injuries seem to play no part in the causation of the disease, neither is there any connection with epilepsy to be traced. Rickets is regarded by some authorities as a most important causal factor; by others as having no connection with the disease. Many of the cases show signs of slight rickets; some exhibit no indications whatever; and the fact has weight that head-nodding seems never to be associated with the well-known neuroses occurring in rickety children—laryngismus, carpo-pedal contractions and convulsion. Dwelling in badly lighted rooms seems to favour the occurrence of the disease. The pathology of this affection is unknown. It is probably of similar causation to congenital nystagmus.

Symptoms.—Children affected with head-nodding are generally very intelligent, often precocious; occasionally mental deterioration comes on as age advances. The most common form of movement is lateral rotation of the head, such as signifies negation. Nodding movements like those of a mandarin doll are rare; sometimes the movements are very complex. In salaam spasm, for example, the head is bent on the breast and the whole trunk is bent forwards. The movements are sometimes constant, sometimes paroxysmal, and always cease when the child is asleep, when he lies down, and when the eyes are covered.

The prognosis is highly satisfactory, for in the majority of recorded cases cessation, both of the head movements and of the nystagmus, has occurred.

Eclampsia nutans is a condition occurring between the time of the first dentition and puberty, and is characterised by peculiar bowing movements of the whole body, so that the head very nearly touches the knees. These salaam convulsions occur in paroxysms, and the paroxysms may occur as frequently as one hundred times a day. Though the subject of this condition may be intelligent before its onset, marked mental failure comes on subsequently and may end in idiocy. Some cases, however, slowly recover.

Since loss of consciousness may accompany each salaam it has been argued that eclampsia nutans is a form of petit mal, but these cases have never been seen to develop epilepsy and have no etiological connection with that disease. It is probable that the condition is a more severe form of head-nodding. The treatment of the above conditions is that for habit spasm.

Spontaneous movements of the head occur in certain other diseases of the nervous system; in Friedreich's disease slow nodding movements are common; the Mongolian type of idiot often

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presents similar movements, and these are also met with in cases of cerebral diplegia and some cases of cerebellar tumour. The nodding tremor of disseminated sclerosis and of paralysis agitans are conditions occurring only in the adult, and are easily distinguishable from the above conditions.

SPASMODIC TORTICOLLIS

Syn. Spasmodic Wry Neck

A functional disease of the nervous system, characterised by tonic or clonic contraction of the superficial and deep muscles of the neck, causing the head to assume either a position in which it is turned to one side and upwards, or a position of marked retraction (retro-collic spasm).

Etiology.—The disease is most frequently met with in middle-aged adults, but it may occur at any age from puberty onwards. It is twice as frequent in females as in males. The causation is most obscure. Not infrequently neuropathic heredity, such as epilepsy and insanity, exists, and the patients are often of highly-strung, nervous, irritable dispositions.

Nervous shock, prolonged anxiety, and general ill-health have frequently preceded the onset of symptoms. Less often local strain, or injury and exposure to cold, have been the presumably exciting causes. In a few cases it appears to develop from an occupation neurosis; it developed for instance in a tailor who in drawing each stitch had the habit of making a short jerking movement of the head to one side. It occasionally occurs as a symptom of hysteria, but such cases should be carefully separated from those in which there is no hysterical manifestation, as being more susceptible to treatment and having no tendency to recur when once cured.

Symptoms.—The onset is usually insidious, but in rare cases may be quite sudden, as in the case of a man aged forty years who, when walking along a London street, suddenly turned his head at the sound of an accident which shocked him severely; he was unable to turn his head back without using his hands to do so, and he subsequently developed the most severe torticollis. The initial symptom is always spasm, which may be either tonic or clonic, and frequently both forms of spasm are combined in the same case.

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In the tonic form the head is retracted and the face turned to one side, usually the left, and, owing to the retraction of the head, the face is turned upwards. The shoulder on the side to which the head is inclined is usually raised. In severe cases all the muscles of the upper extremity, the scaleni, and the face muscles may become involved. The spasm, except in the earliest stages, always involves muscles of both sides of the neck. Where the bilateral involvement is general and equal the rotation of the head does not occur, but it becomes strongly retracted and the condition is then known as retro-collic spasm. Such retro-collic spasm is always accompanied by marked over-action of the frontalis, the skin of the forehead being thrown into transverse wrinkles. In the clonic variety there is jerking movement of the same muscles, usually associated with some degree of tonic spasm. The eyes do not follow the movements of the head in the jerkings.

The muscle primarily involved is the sterno-mastoid, the action of which is to incline the head forwards and towards the shoulder of the same side and rotate the face to the opposite side. The next muscle involved is the splenius of the opposite side, which inclines the head backwards and rotates the face towards its own side, its rotatory action thus coinciding with that of the opposite sterno-mastoid. When the splenii of both sides act together the head is strongly retracted. Next to be affected are the upper part of the trapezii, the trachelo-mastoids and other deep neck muscles, and with further spread of the spasm any neighbouring muscles of the shoulder and upper extremity may be affected. The sterno-mastoid and the trapezius are supplied chiefly by the spinal accessory nerve, and also by the anterior primary branches of the second, third, and fourth cervical nerves. The splenii and the other deep neck muscles are supplied by the posterior primary branches of the upper five cervical nerves.

Sleep causes cessation of the clonic spasm, but not always of the tonic spasm, when the case is severe. The spasm is always increased by fatigue and excitement. There is no wasting of the muscles involved, but on the other hand they may be even hypertrophied if the spasm has existed for long, and their electrical excitability may be increased.

The amount of pain associated with the spasm varies greatly. There may be a slight feeling of cramp only, but usually there is a great deal of aching pain, which may radiate down the arm and into the side of the head, and make life unbearable to the patient. More rarely sharp neuralgic pains are present.

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The course of the disease, which has no tendency to shorten life, is chronic, exacerbations and remissions under treatment being common and recurrence after temporary cure frequent.

Pathology.—No morbid anatomical changes have been found. On account of the involvement of several muscles effecting special movements in this disease (as is well instanced by the over-action of the frontalis in retro-collic spasm, for retraction of the head is always normally associated with raising of the eyebrows in the act of looking up), it is probable that torticollis is due to functional disorder of those cortical centres which direct such associated movements of the affected muscles.

Diagnosis.—This is usually quite simple. Fixed positions of the head associated with spasm occur in disease of the cervical spine, especially in spinal caries, and are also associated with enlarged lymphatic glands in the neck. The local signs of these conditions, however, are characteristic.

Treatment.—Temporary relief is often obtained by the administration of chloral and bromides; keeping the patient constantly asleep for a period of three or four weeks by the administration of chloral hydrate in ten-grain doses administered six-hourly, the patient being meanwhile isolated from noise and disturbance, has produced permanent benefit in several cases in the hands of Dr. Bastian. Morphia always affords temporary relief, but its use is fraught with the danger of the patient acquiring the morphia habit. In one long-standing case one of us (W. S. C.) has known recovery follow deep etherisation repeated on several occasions.

Surgical procedures meet with temporary, sometimes with permanent, success. Tenotomy of the affected muscles rarely gives lasting relief, and the same may be said of the division of the nerves supplying the affected muscles, for such nerves rejoin. The operation of Keen, in which there is extensive exsection of the posterior branches of the spinal nerves supplying the affected muscles and of the spinal accessory nerves, has given encouraging results. Kocher recommends exsection of the whole sterno-mastoid muscle, and Chiene advises extirpation of that cortical centre (No. 12 of Ferrier) which is associated with the movements of lateral rotation of the head.

There is a CONGENITAL FORM OF TORTICOLLIS which is of very different nature. The disease is pre-natal and analogous to congenital talipes, the sterno-mastoid alone is affected, and nearly always that of the right side. Such a muscle is frequently ruptured

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during birth, and this has given rise to the opinion that the birth injury and subsequent hæmatoma of the muscle was responsible for the torticollis.

In many of these cases there is marked facial asymmetry, the face being smaller on the side of the affected sterno-mastoid. This association points strongly to some defect in the nerve centres of the medulla as the cause of congenital torticollis.

The **treatment** of the condition consists in tenotomy of the contracted muscle.

OCCUPATION NEUROSES

This is the name given to a group of diseases in which, after excessive employment of some combination of muscles for particular movements, there is inability to perform that action, and this without impairment of the use of the muscles for other movements. The name is given because the movement affected is usually one required in the daily occupation of the patient. The type of such diseases is writers' cramp, and the description of that troublesome disorder applies, with slight modifications, to the other forms, amongst which telegraphists' cramp, pianists', violinists' and drummers' cramp; typists', machinists' and crochet workers' palsy; hammer and file-makers' palsy; cigar rollers', sempstresses', dairymaids' and dancers' cramp, and miners' nystagmus are the craft palsies which are the best known.

General Etiology.—Craft palsies arise in connection with trades that involve a constant repetition of the same act. While it is certain that increased function of a particular organ of the body leads to increased structural perfection, this is true within a certain limit only, and beyond this limit, according to the rule often known as Edinger's Law, the constant repetition of an act to the point of severe fatigue leads ultimately to atrophy and paralysis of the muscles concerned. No more striking example of such atrophy can be given than that which occurs in the right biceps of the Sheffield file-makers, whose work involves a constantly repeated sharp contraction of this muscle. Again, a faulty position or method in performing an act is a most potent factor in the production of occupation neuroses, for fatigue is then much more quickly produced. For the same reason acts performed in strained

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positions, such as violin playing, are more liable to break down than those performed in a more comfortable attitude.

Local affections of joints, ligaments, and tendon sheaths may, by their interference with the smooth performance of an act, sometimes serve as exciting causes, and for this reason a careful physical examination of these structures should never be omitted.

Impairment of general health, bad hygienic surroundings and mental worry are frequent factors accelerating or allowing of the neuro-muscular breakdown.

Direct inheritance is sometimes traceable, but more frequently some family neuropathic tendency, such as epilepsy or insanity, are present. Parental alcoholism is apparently a factor in some cases.

Writers' Cramp

Syn. Scriveners' Palsy

This complaint is met with chiefly among young adult males whose occupation consists chiefly in writing, particularly among those who adopt a stiff, formal, cramped style of handwriting. Hence a large proportion of the victims are law writers or etchers of copper-plate writing. There appears to be an inherited tendency to the disease, as several cases have occurred in brothers, and not infrequently there is a history of alcoholism, fits, insanity or other neuropathies in the family. Local injury of the hand and mental distress or anxiety are frequent predisposing causes, and the mental anxiety is also aggravated by the disability incidental to the disease.

Symptoms.—The onset of symptoms occurs for the most part in a somewhat definite order. The patient first notices stiffness in performing the act, which is later accompanied by pain or marked discomfort. Afterwards weakness in the affected limb and lastly tremor appear. The commencement is usually insidious. At first there is only an unusual sense of fatigue at the end of the day's work, but before long it troubles the patient almost as soon as he begins to write and compels him to desist. Almost from the first this sense of fatigue is accompanied by characteristic motor and sensory symptoms.

The *motor* symptoms may be spasmodic or paralytic. In the spasmodic form, which is much the commoner, the patient finds that, if he attempts to continue to write after the sense of fatigue has appeared, there are contractions of the fingers, usually accompanied by pain; and that if he still persists the painful

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cramp spreads up the arm and may even involve the shoulder muscles. The same muscles are not affected in all cases. Most frequently there is spasm of the intrinsic muscles of the hand, flexing the digits, which are applied to the pen, and preventing writing by making the pen dig into the paper. In order to circumvent this the patient holds his pen in the fork between thumb and index finger, or that between index and middle fingers, so that the fingers may not be used in writing. This often gives a respite for days or even weeks, but does not avail long, for flexion of the wrist makes its appearance and causes the pen to stick in the paper, and this the patient tries to antagonise by placing the left hand beneath the right wrist to resist the flexion. But again the relief is only temporary, and in time the spasm extends until the whole arm is useless for writing purposes. In other cases the spasm is mainly extensor in character. When cramp occurs the fingers are extended and spread out, so that the pen drops from the hand.

In many cases (about half) the patient is not benefited by writing with his left hand, as when this is learned the spasm commences in that hand also, and if writing is persevered with, the cramp will affect the whole arm.

In the *paralytic* form, which is rare, instead of spasm occurring there is paralysis of the muscles concerned in holding the pen, which usually comes on suddenly, so that the pen falls from the grasp.

In both spasmodic and paralytic forms it must be remembered, as characteristic of the disease, that the hands can be used quite well for other purposes, so that the patient may be able to play on the pianoforte or use a typewriter with comfort, and can perform small movements, such as those of buttoning the clothes, without difficulty or pain.

Atrophy of muscles may occur in severe and in protracted cases. Such atrophy is not confined to individual muscles but affects the hand and fore-arm generally. It is not associated with definite changes in electrical excitability, and, when once marked, the muscles hardly ever regain their previous size.

The *sensory* symptoms are of two kinds: (*a*) Painful cramp, associated with use of the hands for writing, not present at other times. (*b*) General neuralgic pain along the course of the nerve trunks, which may be tender on pressure. It is more or less constant. Occasionally there is a sense of tingling at the tips of the fingers, but there is no anæsthesia on careful testing.

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Electrical reactions.—It is doubtful whether there is any diminution in the reaction of the affected muscles, as has been described by some writers. On the contrary, in cases where neuralgia is a marked symptom, there is often over-excitability to both galvanism and faradism.

Course of the disease.—Unless writing is desisted from at the first appearance of symptoms, the disease is nearly invariably progressive. Ultimately recovery may occur, but it may be reckoned that at least a month of abstinence from writing will be necessary for every week that any symptoms of the disease have been present.

Diagnosis.—In this and in the allied conditions enumerated above a thorough examination of the nervous system is necessary, for this may reveal some congenital deficiency or birth injury or organic disease which may account for the breakdown of function. Again, local disease of joints or synovial membranes may induce a condition simulating a craft palsy or may aggravate one already present. It is not very rare for patients with a slight form of paralysis agitans, in which spasm is preponderant, to inform the physician that their trouble is writers' cramp, for the symptoms of that disease may first obtrude upon the patient in the act of writing.

Pathology.—No morbid changes have been found in connection with the disease, and nothing is known as to its exact pathology. It is certainly an affection of the central nervous system and not a local one of the muscles or nerves.

From the resemblance of the symptoms of craft palsies to those of physiological fatigue in muscle it is possible that a morbid state of the nervous centres for the faulty act, induced by constant overuse, causes the phenomena of fatigue to appear on slight exertion of the centres.

Treatment.—The essential factor in successful treatment is immediate and complete abstinence from using the affected hand for writing. This will be necessary in all cases for about three months. In cases of any standing twelve months or even two years or more will be required. In most cases complete rest of the arm is unnecessary and inadvisable, and the patient may use it in moderation for playing the pianoforte, tennis, etc., and he may profitably employ his time in learning to use a typewriter, as the exercise appears to have a beneficial effect in some cases. A regular course of gymnastic exercises, such as the use of light Indian clubs and dumb-bells, is beneficial in all cases except those where much paralysis is present. If, after the disappearance of symptoms, it is unavoidable that the patient should again return to the same

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occupation, it is all-essential that he shall learn some modification of method in performing the act, so that movements other than those at first in fault shall be called into play.

In the paralytic form regular massage to the affected parts, combined with the daily application of galvanism, is often most useful; but, on the other hand, these measures are of little value in the spasmodic stage. Confinement in a fixed apparatus has been recommended, but is inadvisable. The only cases in which rest of the limb is to be advised are those in which there is much associated neuralgia. Drug treatment appears to have little direct effect, but the sedative effect of the bromides is often most useful in the spasmodic stages. Tonics, such as nux vomica, iron, and cod-liver oil, may be given to improve the general health, and if there is severe neuralgia it must be treated on the same principles as neuralgia elsewhere.

Any local disease of joints, tendon sheaths, etc., which may have an effect in aggravating or keeping up the neurosis must be carefully treated.

PARALYSIS AGITANS

Syn. Parkinson's Disease, Shaking Palsy

A disease of the second half of life, characterised by tremors, usually beginning in the hand, but becoming generalised, together with weakness and afterwards rigidity of the affected muscles.

Etiology. — About two-thirds of the patients are males. Symptoms usually begin between the ages of forty and fifty, and it is rare among younger subjects. Direct inheritance does not appear to play an important part in causation; in only one-sixth of the cases can some neuropathic heredity be traced. The most frequent exciting causes are prolonged grief, worry, or other depressing mental states, and it may follow an accident, in which case the chief factor is probably the mental shock, but in not a few instances the tremor begins on the side which was most injured by a fall or blow.

Symptoms.—In a well-marked case the picture presented by the patient is very characteristic. He shuffles into the room, and walks feebly, with his head and body inclined forwards, his arms held with the joints semiflexed, the hands tremulous, his facial

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expression fixed and, as a rule, indicative of mental depression. Considering the symptoms in detail—

Tremor.—This is nearly always the first symptom. Occasionally rigidity may come on without previous tremor, and indeed tremor may be absent throughout when the other signs of the disease are typical. It usually begins in the hand, sometimes in the foot, and rarely in the face and tongue. The movements are rapid, fine, and usually rhythmical. The most characteristic movement is a rapid action of the thumb against the index finger, similar to that in rolling a cigarette. The movements are for a time controlled if an object be grasped in the hand, in that way differing from the movements in disseminated sclerosis, but in certain severe cases the tremor may be increased on movement, and resemblance to the latter may be very close. The movements continue during rest in waking hours, but rarely persist during sleep. The hand is not long the only part affected, and tremor affects the rest of the arm and then usually appears in the leg of the same side, occasionally showing itself first in the opposite hand. Its distribution therefore is usually hemiplegic, and both the tremor and rigidity may remain confined to one side for years. It may spread to the muscles of the trunk, to the neck muscles, the masseters, the muscles of the tongue, and the facial muscles. It may be said that those parts of the limbs concerned with the highest voluntary movements are most affected by the tremor, for this is always most marked in the small muscles of the hands, whereas the trunk muscles, which are used in a more automatic manner, rarely show tremor but usually present rigidity alone.

It will be convenient in the description of the disease, in which tremor is one of the most striking features, to classify and compare the phenomena which it is customary to include under the term "tremor."

It may be said that tremor is a normal phenomenon, for the muscular contractions which execute willed movements are not continuous, but consist of a fusion of brief muscular contractions, the rate of which varies from eight to forty contractions per second. In conditions of asthenia, muscular fatigue, etc., the rate of muscular discharge becomes slower and less regular; the fusion of the simple muscular contractions which make up the voluntary movement is less complete, and these become apparent in the movement as tremor. Clinically every degree of tremor is met with, from the finest and most regular oscillation to coarse, jerky movement. The finer varieties of tremor, on the one hand, are allied to spasm. This

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relation was first pointed out by Dr. Hughlings Jackson in paralysis agitans, where in some muscles spasm is conspicuous, in others tremor. If we conceive the oscillating movements of this disease increased greatly in rate and the amplitude of the movement diminished, the result will be a condition of muscular rigidity, and such a sequence is the rule in long-standing cases of paralysis agitans. The relation to spasm is true of some varieties of tremor only, for the term includes phenomena which are widely different in nature, and are probably due to defects in very different elements of the nervous system,—for example, the fibrillary tremors which occur after the nerve to a muscle has been severed are probably of muscular origin; the fibrillation which occurs in sciatica is caused by changes in the peripheral nerve; that occurring in progressive muscular atrophy is referable to changes in the anterior horn cells. The tremor of emotion and of general paralysis, on the other hand, are probably of cortical origin. Tremors which occur when the muscles are at rest are for the most part of small range and regular; those occurring on exertion are often of large range and irregular; not infrequently the latter are in reality perverse movements, the results of incoordination. For example, the "intention tremor" of disseminated sclerosis is an incoordinate movement, and allied to such a condition as stammering.

Our knowledge of the physiology of tremor is not sufficient at the present time to allow of a scientific classification. The division into those occurring during rest and those occurring on exertion does not provide for many cases. As an example, the tremors of paralysis agitans are sometimes much increased by exertion, and the tremor of disseminated sclerosis may be conspicuous in the neck and trunk during rest.

The table on p. 361 serves to indicate the nature of the common varieties of tremor and the diseases in which they appear.

Muscular rigidity.—Usually makes its appearance in the parts affected with tremor, but the degree of weakness and of rigidity varies greatly. The more powerful flexors overcome the extensors in the larger joints, but in the muscles moving the smaller joints special results are produced. The fingers are usually semiflexed, with over-extension of the proximal phalanx—interosseal position. In consequence of the stiffness of the facial muscles the face becomes fixed, the wrinkles smoothed out, and there is no play of expression either in emotion or when the patient talks. Owing to this rigidity there is a tendency for tears and saliva to escape. Stiffness of the laryngeal muscles leads to a monotonous mode of

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	Description.	Effect of Rest and Exertion.	Occurrence.
Fibrillary tremor (Myokymia)	Local contraction of muscle fibres occurring irregularly; visible upon the surface covering the muscle, but producing no movement of the limb.	Irrespective of rest or exertion.	Conditions of fatigue; marasmic states; after section of nerve to muscle; interstitial neuritis; progressive muscular atrophy; amyotrophic lateral sclerosis (in wasted muscles only).
Fine tremor .	1. Regular oscillations of quick range, involving antagonistic muscles—never producing a purposive movement.	Generally occurring at rest; often much more apparent during exertion.	Emotion; asthenic states; senility; toxic states; alcohol, lead, zinc, mercury; toxæmic conditions; Graves' disease, etc.; after hemiplegia.
	2. Irregular fine oscillations, often of a twitching character.	On exertion.	General paralysis.
	3. Regular oscillations from fine to coarse, involving a definite group of muscles, and producing rhythmic movement, often purposive in nature, *i.e.* like a voluntary act.	Generally diminished by voluntary movement.	Paralysis agitans; some conditions of degeneration of the cerebral cortex (Weill).
Coarse tremor .	Oscillations irregular in range and time.	As a rule appearing on movement only.	Hysteria; ataxic states; after hemiplegia.
Intention tremor	Coarse irregular jerkings showing a crescendo from the commencement to the attempted completion of a movement.	Do.	Disseminated sclerosis; lesions of superior cerebellar peduncle; lesions of optic thalamus; cerebral diplegia.
Myoclonus .	Shock like twitching, involving from a small part to the whole of a muscle; producing simple movements only, irregular in time.	Uninfluenced by rest or exertion; ceasing during sleep.	Paramyoclonus multiplex, and epileptiforme; cerebral diplegia; (congenital chorea).
(Every gradation between fibrillary tremor and myoclonus is met with clinically)			

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speech, which is hesitating, and, in advanced cases, is also high-pitched. Due to the rigidity of the arms the handwriting becomes small as well as tremulous, and the patient finds it difficult to write in a straight line. The flexor muscles of the body being rigid cause the patient to stoop forward, and in the lower limbs the slight spasticity gives rise to a shuffling gait.

The muscular rigidity is peculiar and often compared to that of lead pipe, the arm being held rigid in any position, and not tending, like an ordinary spastic limb, to return to its former position. If the arm be passively moved, the rigidity passes off for a time. A patient may be so rigid that he is unable to rise from his seat without assistance, and yet can walk fairly freely after taking the first few steps. Not a few cases present sudden attacks of spasm in the feet, the toes being drawn down and doubled into the sole. It usually occurs when the patient is walking without boots, as for instance on getting out of bed. The sudden spasm may cause the patient to tread upon the bent toes, and serious falls have resulted. This symptom was first pointed out by Dr. Purves Stewart.

Attitude.—Owing to the flexor rigidity the body is bent forward and the head is bowed. The centre of gravity is thus brought forward, and when the patient starts to walk he has quickly to take a step forward to support his body in the new position, and hence his gait consists of a rapid succession of shuffling steps. In consequence of the rigidity of the muscles, he cannot quickly call his balancing muscles into action. For this reason a patient may lose his balance in walking, and to save himself from falling he has to run after his centre of gravity, his steps becoming quicker and quicker. This phenomenon is known as "festination." A slight push on the shoulder is enough to make him go forward, and he has to keep moving to prevent a fall. This is better marked if he be pushed back (retropulsion), or even tries to walk backwards. He cannot stop to balance himself, and has to run backwards to prevent his falling on his occiput. A similar phenomenon may occur when he is pushed from the side (lateri-pulsion).

Muscular weakness.—This varies from time to time, and appears to be greater than it really is, owing to the accompanying rigidity. It is usually greater in the upper than the lower limbs. Patients in whom tremor and rigidity are well marked can often walk for miles, the rigidity passing off after the patient has walked a little distance. Ultimately the weakness and rigidity both increase, and the patient becomes crippled and bedridden from the contractures of the trunk and limbs.

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Sensory symptoms.—There are no changes in common or painful sensation, as tested in the ordinary way, but there is sometimes hyperæsthesia to heat. Patients often complain of subjective sensory symptoms, such as formication. But the most common are burning sensations and "hot flushes," which occur chiefly when he gets into bed. There is often also a burning abdominal pain.

Associated with tremor and rigidity there is usually a good deal of aching pain, which is increased by exertion and is not always benefited by rest.

Trophic changes.—In severe cases thinning of the skin with "seeded" nails and some vasomotor palsy is seen in the periphery of the limbs.

Reflexes.—There is no change in deep, superficial, or organic reflexes.

Mental condition.—The patients are usually depressed, but placid and not emotional. Intelligence is preserved unaltered.

Prognosis.—The prognosis is unfavourable, although the patient may live for fifteen or twenty years. The course of the disease is more rapid in the younger patients, and is accelerated by any mental strain or worry.

Diagnosis.—The tremors require to be diagnosed from those of neurasthenia, alcoholism, and disseminated sclerosis. The persistence of the tremor during rest, its regular character and its cessation on voluntary exertion, and the age of the patient will usually make its nature clear. From alcoholism and neurasthenia the accompanying rigidity and the retropulsion will distinguish it, but it must be remembered that paralysis may occur in neurasthenic or alcoholic subjects. By far the most reliable sign in the diagnosis is the general aspect and attitude, at once so characteristic that even a case presenting no tremor is at once recognisable.

Pathological anatomy.—Nothing is known with certainty as to the pathology of this disease, but it is believed to depend on some affection of the pyramidal cells in the motor cortex.

Treatment.—No treatment by drugs appears to influence the progress of the disease, but tonics, especially quinine, add to the patient's sense of well-being. Sleeplessness and pain are best treated with bromides, chloral, and paraldehyde. Mild faradic electricity applied to the skin relieves the paræsthesia, and the abdominal discomfort is usually removed by giving assafœtida in the form of a pill.

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NIGHT TERRORS—PAVOR NOCTURNUS

These are sudden attacks of mental excitement, usually accompanied by hallucinations and always by an intense feeling of fear. They occur in children, and during the first few hours of sleep. Rarely they occur in the waking state (Day Terrors).

These phenomena are always a sign of ill-health in the child. They may occur as occasional events with a definite exciting cause, such as any febrile disturbance, digestive disturbances or peripheral irritation, such as hip disease, etc., and when so occurring have been termed "symptomatic" night terrors. On the other hand, they occur in delicate, nervous, and imaginative children as recurring events, sometimes every night, though rarely more than once in a night, sometimes at longer intervals. They are especially frequent among rheumatic children and those afflicted with nasopharyngeal adenoids. School pressure and nervous excitement are important exciting causes. It is such habitual night terrors that call especially for treatment.

Symptoms.—The children manifesting these symptoms are usually between the ages of three and eight years. The attack occurs almost always within half an hour to two hours of going to sleep. The child starts up with a piercing scream and frequently tries to escape from the room, and if he succeed he runs screaming down the staircase. When found he is possessed with the wildest alarm and clamours for protection, often giving the clue to his hallucination by shrieking "skeletons," "the black dog," "robbers," etc. Though he may cling instinctively for protection to any one, so fully is his attention fixed with the imaginary object of his fear that he does not look at or recognise persons, but may gaze intently at some spot where he locates the object of his fear. The pupils are usually widely dilated, and profuse perspiration is present. The terror resists all attempts at pacification for a period varying from a few moments to half an hour, when the child recognises his surroundings, sinks exhausted, and soon sleeps. There is usually complete recollection of the occurrence, and the dread of its repetition may cause a condition of depression and brooding well calculated to perpetuate the disorder.

The hallucination which starts the terror is most commonly visual, but may be of any kind. Often it is the same hallucination

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which recurs with each attack, and this may be traceable to some picture or object which has frightened the child. In the writer's own experience of night terror, an old chest that he had never seen opened and often wondered on its contents, and a large dog which had once broken his chain and run at him were combined as the constant hallucination; the chest slowly opened, revealing the dog. In older children the hallucination is less vividly remembered, perhaps from being of a more complex nature, and very young children may remember nothing of the occurrence of the terror.

Treatment.—Under careful attention to the child's surroundings, and measures for the improvement of general health, night terrors usually cease in a few months. Drugs are of considerable value. The regular administration of bromides, combined with tonics, for periods of a month at a time, is most useful. Paraldehyde in small doses at night is often useful at the commencement of treatment. Small doses of aloes given regularly have, in many cases, a marked beneficial effect. Cod-liver oil should be given in all cases.

NIGHTMARE.—This condition, which occurs in adults, resembles the night terror in many ways. It is, however, an unpleasant dream which causes the overpowering sense of terror, which lasts until the subject wakes, often with a cry and trembling, but ceases with the waking state. The contents of the dreams of nightmare are varied in the same subject, whereas in night terror the hallucination is constant. Nightmare rarely recurs with a regularity as to require treatment. Mental worries and digestive disturbances are its chief causes.

The treatment consists in regulation of the bowels, avoidance of late meals, and the administration of bromides for a few nights.

SOMNAMBULISM—SLEEP WALKING

A functional disorder which may occur (1) periodically during early life, entirely passing off later; or (2) as an isolated event due to errors of digestion or to the general disturbance associated with the onset of some illness, *e.g.* measles or ordinary sore throat; or (3) as an accompaniment of epileptic fits or unrecognised *petit mal* occurring during sleep. The latter is the most common form after childhood.

During an attack a somnambulist rises from bed without waking,

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and as a rule without putting on his clothes, and makes his way about the room or over the house, or may even go out into the street. His eyes are open and staring straight ahead. He avoids obstacles, though he does not appear to look at them. Accidents have occasionally occurred from patients mistaking a window for a door and walking through it. After some apparently aimless wandering he returns to bed without waking, and next day has not the slightest recollection of his performance. In rare instances somnambulists have been known to perform more complicated acts, such as literary or musical composition.

Treatment.—Attention to the digestive organs, avoidance of excitement and late meals, and the administration of an occasional dose of bromide of potassium at night will usually lead to the cessation of the attacks. During an attack the sleep walker should be led gently back to bed. It is undesirable to awake him roughly.

W. S. COLMAN.
J. S. COLLIER.

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TROPHONEUROSES

AMONG the complex conditions which maintain the nutrition of the highly differentiated tissues of the body, it is usual to reckon the influence of the nervous system, in great measure for reasons that are based upon the observation of certain morbid states. It has already been pointed out (p. 32) that there is no justification for supposing that there are special neurones concerned with the discharge and conveyance of trophic impulses only, but that these are involved in those nervous influences which govern the function of the various organs and tissues, and that "the function and nutrition of these physiological machines form together an inseparable unity." There are therefore no special "centres," nor nerves which are to be regarded as exciting any peculiar trophic influence apart from those by which the functional activity of the several parts is excited and regulated. In a manner which, in contrast to this direct control, may be called indirect and supplementary, is the modification of the blood supply to any region that may be brought about through the vasomotor nervous system; but although a persistent deficiency in blood supply may determine an atrophic or degenerative change in the part so affected, it by no means follows that an excessive supply will of itself lead to any marked nutritional change, hypertrophic or otherwise.

Inasmuch as the nutrition of the body is the outcome of a complex of conditions, some inherent in the living protoplasm, others extrinsic thereto, which are intimately blended in their action, and can scarcely if at all be considered as producing their effect independently of one another, it becomes entirely arbitrary and artificial to arrange any group of diseases as due to perverted trophic influence. In doing so for purposes of convenience of description, it must not be assumed that other nutritional influences may not also be involved, but simply that, so far as can be at present recognised, the functional and structural abnormality appears to be mainly dependent on some imperfection in the nervous control, whether this is exerted directly in stimulating the functional activity of the tissue at fault, or so to say indirectly, as for example by influencing prejudicially the mutual relationship of the tissues, one aspect of which we appreciate as the effect of internal secretions,

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or by depreciating that power of adjustment to the environment which is a fundamental property of bioplasm. Whatever be the exact mode of action, one result is that the resisting power of the tissues is lowered, and deleterious factors of the environment—mechanical, chemical, microbic, etc.—are enabled to produce an effect which a completely healthy tissue would have withstood.

For convenience of consideration, the various pathological conditions comprised within this group may be divided into (i.) those with which a definite nerve lesion is to be found, and (ii.) with which no appreciable alteration in the nerve centres or nerves has yet been constantly detected. Under the latter might be included such maladies as diabetes, gout, obesity, and rheumatoid arthritis, by those who would regard the nervous factor in the causation of these affections as of predominant importance. Nor does such an arrangement exclude the possibility that the determining cause may ultimately be found to be of toxic nature and of microbic origin, producing its damaging effects upon the nervous tissue, and in that way leading to the tissue defect.

(i.) *Trophic lesions following on, or associated with definite structural change in the nervous system.*—These may involve the integuments, the muscles or the bones and joints, in varying degrees of combination, or singly. According to the seat of the disease, whether in brain, in spinal cord, or in the nerves, may the trophic change be local or widespread.

As illustrative of a general trophic perversion may be mentioned the wasting, often rapid and considerable, and far in excess of what may be attributed to disease or deficient food, that is seen in acute mania and to a less degree in many cases of chorea.

(*a*) THE INTEGUMENTS.—Extremely varied are the cutaneous manifestations of perverted neuro-trophic influence, ranging in degree from a simple and transient urticaria to a permanently destructive lesion, such as "perforating ulcer." The nervous lesions with which they are found associated are for the most part very definite, and are located both in the centres and in the peripheral nerves—myelitis, tabes, syringomyelia, neuritis, and injuries of nerves are those most frequently observed.

The least degree of structural change is represented by a simple atrophy of the skin, which becomes thin, dry, and shrunken, either over extensive areas or only in patches or striæ. Should the change involve the hair follicles the hair may fall off, or it may become white; and the nails exhibit white spots, or are ridged, cracked, and brittle. A more marked degree of atrophy is accompanied by

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a thinness and transparency of the skin, with a characteristic smooth and shiny appearance known as "glossy skin," which is frequently tender. This condition is best seen in cases of injury of peripheral nerves, also in neuritis and lesions of the spinal cord. It is sometimes very noticeable in rheumatoid arthritis, a malady for which there is much evidence of a nervous causation.

Certain pigmentary changes have been attributed to nervous influence, such as leucoderma, morphœa, etc., as well as increased deposition of colouring matter. Purpuric eruptions are not infrequent over areas the innervation of which is impaired.

Various forms of thickening, dryness, and roughness of the skin which characterise the affections known as ichthyosis, sclerodermia, etc., are regarded as owing their origin to neurotrophic perversion.

Erythema and eczema are sometimes met with in regions where the nerve supply has been interfered with, and the relationship of urticaria to a nervous lesion is even more probable. Of all skin affections, however, herpes zoster is that which appears best to exemplify the direct control over tissue nutrition exercised by the nervous system. Occurring on the trunk, the eruption is distributed over a zone which more or less completely encircles one-half of the body (very rarely bilaterally symmetrical), following the course of the fibres from a posterior root ganglion, which is found inflamed or the seat of hæmorrhage, to be followed later by sclerosis and degeneration of the peripheral sensory fibres, as well as of the posterior root fibres into the spinal cord. This change in the ganglion may occur as a primary affection analogous, as Dr. Head points out, to an attack of acute anterior poliomyelitis, the cells of the ganglia being the "morphological equivalents of the large cells of the anterior horn." Or the herpes may be a symptom developed in the course of myelitis or tabes or general paralysis of the insane. An eruption similar in all respects may arise over the course of the ophthalmic division of the trifacial nerve, and changes in the Gasserian ganglion, corresponding to those seen in the posterior root ganglia, have been found in such cases. The dependence of the tissue change which constitutes the herpetic rash, on a distinctly recognisable nervous lesion, appears to be undoubted, and vesicles and bullæ have been described over the course of nerves, the trunk of which has been injured.

Various destructive lesions of the integuments, involving also the deeper structures, are met with in the course of gross changes in the nervous centres or in the nerves themselves. The most important of these are gangrene; bed-sore, which is specially prone

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to follow on diseases of the spinal cord, such as transverse myelitis; perforating ulcer, usually of the foot, a not infrequent occurrence in tabes dorsalis, and much less often in syringomyelia, spina bifida, Friedreich's disease and peripheral neuritis, most frequently of the diabetic variety; and a painless form of whitlow, which forms on the ends of the fingers in that form of syringomyelia known as Morvan's disease. Many of these conditions are undoubtedly determined by injuries often so trifling as to produce no effect in healthy subjects, but sufficient to bring about extensive destruction in the ill-resisting tissues whose innervation has been impaired, and particularly so if there be any anæsthesia which diminishes the protection of voluntary avoidance. Very often, however, no external cause is apparent. It is further to be remembered that, whatever may be the share respectively of mechanical injury (be that no greater than pressure by the bed) and of perverted trophic influence, a great part of the resulting destructive effects is due to microbic infection, which would be successfully resisted by the healthy skin.

As a further illustration of a morbid trophoneurosis, leprosy may be adduced. In this disease the nerves and spinal ganglia, among other structures, are invaded by a specific bacillus, and a neuritis is set up, leading to various changes in the integuments, mainly of a destructive character, in which the deeper tissues, even to the bones, are involved. Doubtless these results are in part brought about by the presence of the organisms in the skin, but some appear to be as certainly caused by the damaged innervation which the microbes have also induced. (See Vol. I. p. 235.)

(*b*) THE MUSCLES.—Nutritive changes in the muscles, represented by atrophy, granular and fatty degeneration, with fibroid substitution, are of frequent and characteristic occurrence in various diseases of the nerves and of the nerve centres, especially in lesions of the cells of the anterior cornua and their homologues in the medulla. In such maladies as acute and chronic anterior poliomyelitis, and amyotrophic lateral sclerosis, a wasting of the muscles is met with far exceeding what may be accounted for by disuse. In these diseases also the trophic disturbance is chiefly manifested by the muscles, the skin suffering but little or not at all, and only in infantile paralysis is any change to be found in the bones. Muscular degeneration resulting from toxic neuritis is seen in the paralysis of palate, heart, diaphragm, etc., met with in diphtheria. Electrical examination shows the "reaction of degeneration." In attributing these muscular changes to perverted trophic influence, as is usually done, it should be remembered that exactly similar or even more

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advanced changes are to be found in the so-called primary or idiopathic myopathies, in which no lesion of centres or nerves has hitherto been discovered.

The comparatively rare affection known as FACIAL HEMIATROPHY has been commonly regarded as a typical illustration of nutritive change in tissues due to perverted nervous influence. The disease, which commences in early life, usually before puberty, and more often in females, is at first characterised by the skin over one side of the face becoming thin and glossy with atrophy of the papillary layer of the corium; there is also a shrinking of the integuments from absorption of the subcutaneous fat. Later on a wasting of the muscles, sometimes involving the tongue, and of the facial bones, sets in. The hair is also likely to fall out and the cutaneous secretions to be much diminished. In this way very considerable deformity may be brought about. It is to be noticed, however, that although the skin changes are of a character commonly referred to disturbed trophic governance, the muscles do not exhibit the reaction of degeneration and readily respond to the will or to emotion. Sensation is not affected. So far as the very scanty post-mortem records show, an interstitial neuritis of the trifacial nerve is to be found with "atrophy of the so-called descending trigeminal root," which has been shown to be motor in function. If the affection be due to impaired trophic influence excited by the trifacial, it is difficult, as Dr. W. A. Turner has pointed out, to explain how, with such a profound skin change, sensation is unimpaired.[1] To refer the disease, as he does, to "an arrest of development during the growing period or towards the end of it" scarcely advances the explanation, nor indeed does such a view exclude it from being a trophoneurosis, due to some deeper lesion than trigeminal neuritis. In seeking for a cause, the extremely rare condition of unilateral facial hypertrophy that has been known to set in after puberty must be considered, as also the diseases known as acromegaly, gigantism, and the like. It may ultimately be proved that the responsibility for these formative aberrations is to be found inherent in the tissue elements themselves, rather than in any influence, trophic or vascular, that may be brought to bear on them (see p. 237).

(*c*) THE BONES AND JOINTS.—Those structural diseases of the brain and cord which lead to paralysis are likely in time to produce an atrophy of the bones, sufficient, occasionally, to permit of their spontaneous fracture. Such a change, however, may

[1] Allbutt's *System of Medicine*, vol. vi. p. 487.

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probably be attributed to mere disuse and the impaired nutritive changes implied therein. Should the primary lesion occur in early life, before full growth has been attained, the degree of osseous atrophy may be very considerable, and contribute in great measure to the general ill-development of the affected region. Such a condition is best seen in cases of acute anterior poliomyelitis, when the coincident muscular atrophy renders the limb more or less completely useless and imperfectly formed, the loss of muscular power removing that stimulus to healthy nutrition by which the structural development and integrity of the tissues is determined and maintained. A similar atrophy of the bones of the extremities from lesions of the peripheral nerves is to be seen in leprosy.

Injury or compression of nerves has been known in rare cases to be followed by changes in the joints—usually those of the hands and feet. They exhibit moderate swelling, redness, and a variable degree of pain, and finally become stiff from fibrous ankylosis. The exact relation of these phenomena to nervous influence is a matter of debate. Even less certain is the connection in the alleged cases of arthritic changes associated with hemiplegia of cerebral origin.

Trophic changes in the bones of a more profound character, and more distinctly attributable to impaired nervous influence, are to be seen occasionally in locomotor ataxy, and even more frequently in syringomyelia and chronic insanity. The change essentially consists in a decalcification and absorption of the osseous tissue, whereby a "rarefaction" of the bone is brought about, with enlargement of the Haversian canals and medullary cavity, in which the epiphysis no less than the shaft is involved. The long bones of the limbs are those most commonly affected, but the short bones of the extremities, the vertebræ, or indeed any part of the skeleton may exhibit the alteration. Fatty degeneration, with a round-celled infiltration in the medulla, usually coexist.

As a result of this alteration in the structure of the bones, they tend to break with extreme readiness, and in consequence of trifling injury, or even by the normal contraction of the muscles attached to them. Yet such fractures as a rule, though not always, heal with the formation of considerable callus. Should the spine have fractured, much deformity may follow. It is noticeable that there is usually little or no pain connected with these fractures, though in tabetic cases it is said that they more frequently occur in those limbs which have been the seat of marked "lightning pains."

Still more striking are the changes which may be met with in

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the joints in these two maladies. In tabes the knee and hip are by far the most frequently affected, rarely the upper limb articulations, unless in very advanced cases; whilst in syringomyelia the shoulder, elbow, and occasionally the wrist are those which are usually involved. Sometimes several joints are disorganised, and sometimes in tabes the incidence of the condition is symmetrical.

It is seldom that the affected joint exhibits any indication of the change which is about to take place in it, although occasional attacks of pain may be complained of, and it often happens that the actual invasion of the attack is quite sudden; or it may be that some injury, however slight, has precipitated it. Owing to a rapid effusion of fluid into the articulation, great swelling occurs, which may extend far beyond the limits of the joint proper. There is a remarkable absence of pain, a character which prevails throughout and constitutes one of the most singular features of the affection. The swelling may last for weeks or months and then subside more or less completely, when further changes in the part will be found to have taken place. Prominent among these is a certain degree of atrophy of the articular ends of the bones, with which is associated the rarefaction already described; but whilst there is this wasting of the normal osseous structures, there is at the same time a variable amount of new bone formation in the ligaments, tendons, and other structures around the joints, and of bony outgrowths from the ends of the bones themselves. Much variety obtains in the proportion to which the atrophy and overgrowth respectively prevail in any joint, but, speaking generally, the knee and elbow tend to present the greatest degree of new formation along with atrophy, whilst the latter alone, or almost alone, characterises the shoulder and hip. Consequent on this difference is the extent of mobility in the joint, for whilst in the elbow and knee the range of motion may be extremely limited, even to becoming fixed from ankylosis, an undue freedom may be found in the shoulder or hip. Crepitus and crackling and loose pieces of bone can usually be elicited on manipulating the joints, which, partly from fluid and partly from the osseous growth, may be very considerably enlarged and completely disorganised, presenting in its painlessness a unique condition that contrasts with another arthritic disease—rheumatoid arthritis—which in some of its anatomical characters exhibits many points of resemblance to that now under consideration. The extent to which the changes here described—known as "Charcot's joint," since he first gave any account of them in 1868—may occur, varies within wide limits from a mere effusion, that after absorption leaves

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a flaccid joint, to an extreme degree of destruction with complete uselessness of the articulation. These combined osseous and arthritic changes, when occurring in the small bones of the foot, bring about a characteristic deformity—"tabetic foot"—due to the atrophy and yielding character of the bones and disorganisation of the joints, which in an extreme degree may even permit the malleoli coming in contact with the ground, the foot being much shortened and swollen posteriorly. The skin may be thin and shiny, or hard and thickened (sclerodermia), and all degrees of neurosis may be superadded from the presence of a perforating ulcer.

Looking to the similarity of the trophic changes in these two diseases, which in their other clinical manifestations exhibit marked differences, and also to the fact that the only lesion in the nerve centres common to the two maladies involves the gray matter of the posterior commissure of the cord, and of one or both posterior cornua, it is in this situation that the trophic control is regarded as being located, the conducting fibres "either accompanying or are identical with those which conduct impressions of pain or temperature" (Dr. Turner, Prof. Allbutt's *System of Medicine*, vol. vi. p. 559). There is some reason to think that the essential and primary trophic perversion is the atrophic change in the bones to which the joint changes have been supposed to be secondary, being induced by fracture of the rarefied epiphysis. But although it is quite likely that hypertrophy and new growth may in some cases be the expression of a perverted trophic influence, it is not easy to explain the association in the same bone or joint of both atrophy and hypertrophy as being due to the same trophic disturbance. It has been sought to attribute the latter to secondary local irritation, and the thickened callus which forms after fracture of such bones is pointed to in support of the view.

(ii.) *Lesions regarded as neurotrophic, but without constantly recognised disease of the nervous structures.*—It is clear that such a group must be quite artificial, and it is probable that improved and more extended observations will result in relegating many of the diseases about to be described to the previous category.

ANOREXIA NERVOSA

This condition, though not unknown previously, was first described and named by the late Sir Wm. Gull (*Tr. Clin. Soc.* 1874, vol. vii.).

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It occurs most commonly in girls from fourteen to twenty years of age, but it has been met with both in earlier and later life, and occasionally in boys. The most characteristic feature is an extreme emaciation, which comes on during several months, and is unaccompanied by any evidence of structural disease, such as tuberculosis, diabetes, malignant disease, or some forms of cerebral disease. The wasting is profound, far beyond what would be attained by voluntary or enforced starvation of a healthy person, as seen in "fasting men" or shipwrecked persons deprived of food; and it commonly exceeds that seen in any of the wasting diseases mentioned, in which the emaciation is in great measure proportional to the inability to take food. A well-grown young woman may be reduced to five stones or even less in weight.

As the name implies, there is a want of appetite, and the distaste or even disgust for food generally, and particularly for meat, is no doubt in great measure responsible for the condition. It does not follow, however, as has been supposed, that the anorexia is dependent upon some affection of the terminal branches of the vagus in the stomach, but rather is it to be attributed to some abnormal state of the central nervous system which leads not only to impaired appetite, but also to a grave deterioration of the nutritive capacity of the tissues. How distinctly mental or emotional states affect the appetite is well known; but it may be observed that the abstinence from food in this affection is not always so complete as is supposed. The most marked case seen by the writer—a girl aged thirteen years—was in the habit of surreptitiously providing herself with biscuits, cakes, etc., which she went so far as to steal, but, notwithstanding this, the emaciation was progressive and extreme previous to coming under treatment.

Although the loss of flesh may be so great, the patients frequently manifest a constant restlessness, leading to considerable exertion, and the extent of their muscular effort may be surprising, having regard to their appearance.

Among other symptoms which are commonly observed in these cases are a moderate degree of anæmia, a subnormal temperature, a small, infrequent pulse, and amenorrhœa. Sometimes there is vomiting, perhaps after everything that is taken, or there may be diarrhœa. It is usual also to find more or less change in the psychical state; the girl becomes irritable and peevish, or morose and silent, quite different to her normal temperament, and sometimes untruthful and mischievous even to stealing. The line between some of these cases and hysteria is not always easy to draw.

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Short of such severe cases a much less degree of the same state comes within general experience. Young and middle-aged women of a neurotic habit are frequently met with, who, with the expenditure of much physical and mental energy in the pursuit of some philanthropic or similar object, live upon a minimum amount of food, and the same may be seen among religious ascetics, who, by habit, have subdued their appetites and yet accomplish much. In such the loss of bulk may be mainly or entirely due to the lack of food, and may serve to illustrate the important influence of habit in respect to the necessary amount of the ingesta, though it is not to be forgotten that considerable obesity is consistent with a very limited diet. In the more severe cases, however, to which the name *anorexia nervosa* has been specially applied, it would seem that the nutritive capabilities of the tissues are at fault, and that this, as well as the starvation, is responsible for the extreme emaciation.

Although a fatal result of this state is not unknown, a case, however far advanced, may be reasonably expected to recover—care having been taken to exclude the existence of diabetes or of structural disease—provided a particular line of treatment be thoroughly carried out.

This consists essentially in absolute isolation from all friends for six or eight weeks, and for the greater part of the time strict confinement to bed, a liberal diet, and massage. To ensure this the services of a competent, firm, but kind nurse must be secured, who should give her entire attention to the case, and preferably sleep in the patient's room. All communication with her friends by letter or otherwise must be forbidden, and she should not be allowed to read or work, or even to talk, until a definite improvement has set in. The massage should be applied to the whole body and limbs, and, commencing with a quarter of an hour night and morning, should be daily increased until in a week the duration should be an hour twice a day, always at the same times.

The feeding is usually the difficulty, especially at first, and it is well to begin with 4 to 6 ozs. of milk every three hours, increasing it until 3½ to 4 pints are taken in the twenty-four hours. This especially tries the capability of the nurse, who must insist on the quantity being consumed. Within three or four days, eggs, Benger's food, or Plasmon should be added to the milk, and in that way the amount of nutriment is steadily increased. The object to be attained is to make the patient take three good meals a day, supplemented by two or three pints of milk. It is a case of over-

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feeding, and the sooner this is accomplished the sooner will the patient improve, and, as a rule, after the first difficulty is overcome the food is taken readily. The worst cases, *i.e.* those in whom the wasting is most profound, are frequently those who do best. Persons who have by long habit lived upon very little, and have yet accomplished a fair amount of work, muscular and mental, are not so easy to alter in their mode of living, and when the hysterical symptoms are marked the prognosis is not so good. A laxative to keep the bowels regular is all that is required, and it is seldom necessary to give iron or other tonics. It is rare that a case relapses; the cure is generally complete and lasting.

RAYNAUD'S DISEASE

The morbid changes comprised within this term are characterised by a profound alteration in the vascular condition of certain regions, presumably due to spasmodic contraction of the arterioles, causing pallor (*local syncope*), cyanosis (*local asphyxia*), and gangrene. The phenomena are for the most part paroxysmal, and tend to be symmetrical. They are mainly manifest in the hands and feet, less often in the ears and nose, and are frequently associated with symptoms that suggest the existence of a similar vascular state in internal parts.

Etiology—*Age and sex.*—The disease has been met with at almost all ages, as early as 2½ years and as late as 60. Of 43 cases the average age was 26½ years. It is certainly frequent in early life. The same cases showed a preponderance among females.

Hereditary predisposition.—It appears to be associated with a neurotic temperament, and specially to occur in neurasthenic subjects, or in the children of hysterical or emotional parents, who sometimes have actually suffered from one or other of the manifestations of the disease itself.

Previous illnesses.—A sufficient number of cases have occurred in those who have suffered previously from malaria to suggest some causal connection, though what the nature of this may be, if it exists, is unknown. Sir T. Barlow (Prof. Allbutt's *System of Medicine*, vol. vi. p. 603) suggests "that ague may bring about some change in the economy, in consequence of which the vasomotor control or vasomotor resistance may be lessened, and the

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influence of external cold becomes thereby a more powerful factor than under normal circumstances."

The disease has been met with in association with inherited syphilis, and also with diabetes, but to what extent these are to be regarded as predisposing causes is doubtful.

Undoubtedly the most important *exciting cause* of the malady is *cold*. By far the greatest number of attacks come on after exposure, the morning bath being a frequent determinant. Yet some at least of the phenomena of the affection occur in warm or even hot weather, on the slightest fall in the temperature.

As provoking a paroxysm *emotion* or *mental shock* holds a subsidiary though apparently a distinct place, and some outbreaks are apparently traceable to gastric disturbance.

Symptomatology.—The clinical phenomena of Raynaud's disease are separable into three well-marked groups, referable doubtless to the same underlying condition, but readily distinguishable in their character and appearance. These three symptom-groups, known as (i.) Local syncope, (ii.) Local asphyxia, and (iii.) Symmetrical gangrene, occasionally occur successively; more often the first or second occur alone, though either may terminate in the third, though not necessarily so; but they are both extremely liable to recur, the paroxysms being sometimes quite periodic in their incidence. The close resemblance of these conditions to those induced by varying degrees of cold, from mere paleness and blueness to frost-bite, is obvious.

(i.) Local Syncope.—This condition is popularly known as "dead fingers," and consists in one or more of the fingers or toes becoming extremely pallid and bloodless, with shrinking and wrinkling of the integuments, which assume a parchment-like appearance, are intensely cold to touch, with diminution in tactile sensibility, and some slight pain, though this is occasionally severe. Some difficulty in the movements of the affected regions is commonly met with. The condition is symmetrical in invasion, and all four extremities may be involved; and it may extend to the wrist or ankle. It is noticeable that the radial pulse often appears to be quite normal, notwithstanding the obstruction offered to the blood flow by the contracted arterioles and capillaries; should any structural change have occurred in the radial artery, such as arteritis or atheroma, the character of the pulse due to these conditions will, of course, persist during the attack.

All degrees of severity and duration are to be met with, from a slight transient pallor to absolute bloodlessness, lasting for hours and

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repeated for days, and then an interval of relief, the alternation occurring with remarkable regularity. The general health remains unimpaired, and the condition is a purely local one, no general symptoms developing.

(ii.) Local Asphyxia.—The term asphyxia is as inappropriate as syncope in application to the several phases of the malady. By it is meant a sudden discoloration, varying from a moderate duskiness to an intense purplish black, of the upper and lower extremities, limited to one or two corresponding fingers or toes, or involving the entire hands or feet, with lower part of arms or legs. The attack may be preceded by a slight and passing pallor, or it may supervene on the syncope first described. There is but slight swelling of the affected part, but the superficial veins are distended. The ear, nose, and even the face and trunk in symmetrical patches may be similarly affected, though far less often. Except for the colour, the local asphyxia closely resembles local syncope in its effects. Like the latter, the region feels very cold and its temperature falls considerably, the pain experienced being often very severe. In duration, liability to periodic recurrence, and absence of general symptoms, the paroxysms accord with those of syncope.

The condition now described is similar in appearance to chilblains, although the asphyxia affects the extreme tips of the fingers rather than areas short of the ends, and there is not the paroxysmal character which marks the latter state. In chilblains also there is something beyond a mere vascular alteration, there is some degree of inflammation.

These two states of vascular disturbance severally return to the normal, either gradually or almost suddenly. As the circulation is re-established the skin becomes hot, with a sensation of burning and often great pain, with sometimes perspiration as the part acquires its normal colour. In the more severe cases the intervals between the attacks become progressively shorter, until after a few years or more the condition becomes permanent, and the next state, that of gangrene, is liable to supervene.

(iii.) Symmetrical Gangrene.—This is the final stage to which the preceding ones tend, their liability so to terminate being in proportion to their severity. The gangrene is chiefly of the dry variety, and as a rule the bones escape necrosis. Seldom more than the ends of the digits are involved, although extensive destruction of the limbs has been known, and in children rapidly fatal cases of multiple patches of gangrene of the trunk or extremities have been

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seen. After separation of the necrosed portions the ends of the digits cicatrise.

The quite as frequent involvement of the upper limb as of the lower, its more superficial character, and its strict limitation to the region at first affected without spreading, serve to distinguish this condition from senile forms of gangrene due to arterial obstruction, or from diabetic gangrene.

Associated with these external manifestations of vascular disturbance there occur, with sufficient frequency as irresistibly to suggest a distinct connection, certain symptoms referable to internal structures and similarly best to be explained by some vasomotor irregularity. Of these the most important is

PAROXYSMAL HÆMOGLOBINURIA.—The connection of this condition with Raynaud's disease, though long known as an independent affection, is a matter of comparatively recent recognition, and the closeness of the relationship is still a subject of debate. Some would consider the two states as essentially identical; others regard the urinary symptom as but an occasional manifestation of the other malady, and liable to occur in the course of different morbid conditions, such as malaria or syphilis (see Vol. II. pp. 314, 374). Examination of the blood drawn from the region which is the seat of local syncope or asphyxia shows the corpuscles indisposed to form rouleaux, and much crenated and shrunken, whilst the serum contains hæmoglobin. That hæmoglobinuria should occur in such a condition of the blood is obvious, and that the phenomenon is paroxysmal, often extending over years, and is constantly associated with more or less well-marked evidences of Raynaud's disease in the extremities, is certain. An attempt has been made to make the connection closer by regarding the damage to the red corpuscles as being dependent on spasm of the arterioles mechanically injuring those which are less resistant. Nausea, vomiting, yawning, epigastric pain or discomfort have been noted in connection with this symptom, not infrequently preceding it.

Hæmorrhages, such as epistaxis and menorrhagia, have been occasionally met with in the course of the malady, and jaundice has been observed to follow the attacks.

Among the less frequent associated symptoms are certain skin affections, especially urticaria and generalised sclerodermia, which are generally held to be of a nervous causation; effusion into joints, both large and small, and subsequently fibrous ankylosis of the latter; cerebral symptoms in the nature of delusions; partial loss of consciousness or even epileptic seizures; and dimness of

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vision, with contraction of the retinal arteries and pulsation of the veins, and in one recorded case characteristic symptoms were accompanied by iridoplegia.

The pathology of the malady is admittedly obscure, for whilst peripheral gangrene more or less symmetrical may very certainly follow on peripheral neuritis, syringomyelia, or obstructive arteritis, an essential feature of Raynaud's disease is that the local syncope, the asphyxia, and in some measure the gangrene are recurrent and paroxysmal in character, occurring without any evidence of nervous or arterial change, the affected regions in the intervals between the attacks being almost or quite normal. So far as present knowledge goes, definite and severe cases of Raynaud's disease may occur without any recognisable change in the nerves or vessels, yet various degrees of the characteristic phenomena have been found associated with peripheral neuritis. In long-standing cases, Sir T. Barlow thinks obliterative arteritis may exist when the symptoms become persistent. Spasm of the vessels is the only condition that can be definitely affirmed to exist, although the arteries of the affected region during the paroxysm can generally be felt to pulsate normally. At present the spasm must be regarded as dependent on an excessive irritability of the vasomotor centres reflexly excited by cold. The similarity of the gangrene to that induced by ergot is noticeable, and the symmetrical distribution of the disease has been regarded as strong evidence of its essentially toxic origin.

Treatment.—Beyond keeping the extremities warm during the attack and avoiding cold generally little can be done. The pain may be so severe as to require morphia; but otherwise drugs are of little avail, even nitroglycerine, nitrite of amyl, and similar remedies rarely lead to any improvement, so firmly is the morbid habit usually established. Some benefit has been claimed for passive exercise of the limbs, massage, etc., regularly administered over a long period in the intervals between the attacks, and still more for galvanism, the limb being immersed during the paroxysm in tepid salt water in which one electrode is placed, the other being applied to the upper part of the limb. It is recommended that the current should be frequently made and broken, "so as to get repeated moderate contractions of the limbs" (Barlow, *loc. cit.*), the patient meanwhile moving the digits. The gangrene should be dealt with on surgical principles.

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ERYTHROMELALGIA

The essential features of this somewhat rare malady are, as the name implies, pain and redness of the limbs, which are increased when the affected part is in a dependent position. To these symptoms, which are often symmetrical, may be added increased heat, and sometimes swelling of the painful region, the whole occurring in attacks of variable duration, but as a rule repeated over many years, with an inclination to abatement in severity as they become more persistent.

Although clinically presenting a marked contrast in many respects to Raynaud's disease, it appears to be probable that these maladies are allied in their nature, and cases presenting symptoms intermediate between the two are occasionally met with.

Etiology.—Most of the cases recorded occurred in men in early middle life; it is very rare in children.

Exposure to changes of temperature and heavy work have been usually regarded as determining a liability to the disease, but it has been met with in patients living under quite opposite conditions. Various infective diseases have been credited with predisposing to the malady, such as malaria and gonorrhœa. There appears to be no special prevalence among neurotic individuals.

Symptoms.—These appear oftener in the feet than in the hands, and usually in one extremity first, the other being later affected. Very rarely is the face or trunk involved. In many cases, pain of a throbbing, burning character in the soles or palms is the earliest symptom, and may be the only one in the first attacks, or its appearance may coincide with that of the vascular disturbance. When the region affected is analgesic, tingling rather than pain is experienced. The symptom is readily induced or intensified by the dependent position of the limb, and often by warmth, exertion, or even emotion. Sooner or later the painful area becomes a bright red, and subsequently assumes a deep purple-red, the veins being prominent, and the whole region being felt to pulsate. The skin is tense and shiny, and during attacks is often covered with perspiration. The occurrence of the "vascular crisis" is favoured by the same conditions as determine the pain, and similarly is relieved by elevating the limb or by cold. The surface temperature of the region is raised when the limb hangs down, and falls on its being elevated, which is the opposite to that which obtains in a

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normal condition. Some swelling is often to be seen, and hyperæsthesia of the part is frequent. Atrophic changes in the skin and nails are sometimes met with. Hæmoglobinuria is not associated with the disease.

The paroxysm generally lasts three to four hours, but it may be of much shorter duration, or even extend to days or weeks. After the condition has been established for some time, the attacks set in spontaneously, and without any such determining causes as position or warmth.

Pathology.—As with Raynaud's disease, the intimate nature of erythromelalgia is very little understood, though there is much reason to regard both as expressions of vasomotor ataxia. Yet notwithstanding the apparent existence of the malady apart from any detectable structural change in vessels or nerves, there is abundant evidence to show that erythromelalgia is a not infrequent accompaniment of such definite lesions of the spinal cord as tabes, disseminated sclerosis, syringomyelia, and myelitis,[1] or even more frequently of peripheral neuritis. The oftentimes sharply limited area involved, independently of nerve distribution, has been adduced as an argument in favour of the vasomotor nature of the affection. At the same time oft-repeated vasomotor disturbances may lead to degeneration and other changes in the walls of the vessels involved. More knowledge as to the exact condition of nerves, nerve centres, and vessels is required in respect to both these maladies before their pathology can be regarded as understood. Dr. Collier (*loc. cit.*) remarks—"In several of my cases there occurred at first only spontaneous attacks; afterwards the condition became frequently induced by the dependent posture, and later a condition of permanent vasomotor paralysis of greater or less degree made its appearance, the attacks meanwhile continuing. This sequence suggests an irritative lesion of nerve structures governing the blood-vessels being the cause of the vascular crisis, and of the progress of this irritative lesion to a partially destructive lesion being the cause of the persistent vasomotor palsy, these phenomena in vasomotor nerve elements being parallel with pain followed by anæsthesia in sensory nerve elements and with spasm followed by motor paralysis in vasomotor nerve elements. . . . The term 'vascular crisis' would, I think, be very apt, occurring as it does in tabes associated with gastric and other sensory 'crises.' Probably the

[1] See an interesting series of ten cases, collected at the National Hospital for the Paralysed and Epileptic by Dr. Collier, and published in the *Lancet*, 13th August 1898. Of these, six were females, the ages ranging from twenty to fifty-two years.

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same fundamental pathological processes underlie both sensory and vascular crises. In all my cases the vascular change was never preceded by the sensory disturbance, but either preceded it, or the two appeared simultaneously. It seemed as if the sensory disturbance were a local result of the altered vascular condition of the part. I would lay stress on the fact that erythromelalgia may be the first symptom of organic disease of the cord, and may be of great value in diagnosis, and especially valuable in the differential diagnosis between functional disease and disseminated sclerosis." Although the manifestations of the disease are most commonly localised to the feet and hands, there are not wanting indications of a wider disturbance—face, neck, back, and testis have all been known to exhibit a painful congestion with some swelling, suggestive of an affection of the vasomotor centres which may be stimulated to action either centrally or in a reflex manner.

Treatment.—The very distinct relief by elevating the affected part, and by keeping it cool and as far as possible free from pressure, obviously suggest the mode of treatment of an attack. Faradism or massage have been sometimes found of use, and the pain may be so severe as to require morphia for its relief. Very little can be done when the condition becomes more persistent, nor are preventive measures of benefit; the unsatisfactory results of amputation of the extremity or excision of a piece of the nerve to the part do not encourage further attempts of the kind.

ANGIO-NEUROTIC ŒDEMA

Under this term are provisionally included the various conditions of localised, or rarely general, œdema for which no explanation is to be found in the presence of inflammation, of gross nervous obstruction, or of obvious alteration in the quality of the blood, but which are usually associated with some "nervous" state, and are, for the most part, sudden in onset and transient in duration.

Clinically there is manifested a circumscribed swelling of some part of the body, lips, face, hand, foot, etc., which appears without apparent cause and disappears after lasting a few hours or days. A distinct periodicity has been noted in some cases, and also an occurrence in several members of a family and in several generations. The swelling may be quite free from itching, pain, or other sensation than that of fulness, or it may be red and tingling. Mild gastro-intestinal crises have been noted, and also hæmoglobinuria in exceptional

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cases, suggestive of vascular changes in the viscera. Œdema of the glottis with fatal result has been recorded in such cases.

The pathology of this manifestation, like that of the allied states already considered, viz. Raynaud's disease and erythromelalgia, is most obscure. It would appear to be dependent on some temporary alteration in the condition of the blood-vessels in certain areas, involving a modification in their permeability to the blood serum. Until the precise structural changes which take place are precisely known, and also the conditions governing the transfusion of fluid through the capillary walls, the explanation must remain hypothetical. It is, however, impossible to overlook the resemblance of these localised œdemas to various forms of urticaria and erythema, nor to exclude from the purview those cutaneous eruptions which owe their development to poisons.

The symptom rarely calls for **treatment**, which can only be empirical. The course and duration of the œdema is rarely controlled by drugs, though a calomel purge is frequently indicated.

W. H. ALLCHIN.

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MEDICAL OPHTHALMOLOGY

THE PUPIL

The pupil is the aperture through which light is transmitted to the back of the eye. It is situated a little to the nasal side of the middle of the iris, and in health is round. There is no standard size for the pupil, even in the individual, and it varies with the conditions to be presently alluded to. The two pupils as a rule are equal. Inequality, however, is sometimes present, and is quite compatible with health, although, according to some, it occasionally indicates a neurotic tendency. Contraction of the pupil depends upon the contraction of a muscular band on the inner side of the iris—the *sphincter pupillæ*; dilatation depends on a *dilator pupillæ* muscle, and inhibition of the sphincter. Experiments on animals would seem to indicate that there is also some dilatation effected through the natural resiliency of the iris itself.

Contraction of the pupil occurs on exposure to light and also during accommodation for near vision. Contraction to light is a reflex action. The centripetal fibres for this action run in the optic nerve, while the centrifugal or efferent fibres run in the third nerve. There is considerable difference of opinion as to the actual path which the impulses follow. The most generally accepted view is that the fibres pass with the optic nerve, partially decussate at the chiasma, pass along the optic tract and go to the corpora quadrigemina and thence to the part of the third nucleus subserving the pupil contraction. Thence the path is down the third nerve and by the short root to the ciliary ganglion, the short ciliary nerves and the sphincter iridis. There is contraction not only in the illuminated eye but also a consensual contraction in the other eye. This is effected chiefly through the decussation of the fibres, although there is also probably a direct connection between the two third nerve nuclei. It is believed by some that both geniculate bodies come into the afferent path: others hold that the afferent fibres do not cross at the chiasma, while others believe that the ganglion habenulæ is the centre for the pupil reflex in animals. The pupil also contracts during the act of accommodation for near vision. This no doubt depends upon convergence to a great extent, and it is probable that there is a common centre for convergence, accommodation, and pupil contraction. In dogs it has been found that in the posterior part of the third ventricle the centre for the ciliary muscle, the sphincter pupillæ, and the internal rectus muscle are placed in close succession.

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Dilatation of the pupil is probably the result in large measure of stimulation of the cervical sympathetic. There is thus induced an inhibition of the sphincter pupillæ, and the posterior limiting membrane of the iris probably aids by its elasticity in the dilatation. According to the latest researches there is a dilator pupillæ muscle. There must, also, be some dilating power inherent in the iris itself, for when from paralysis of the third nerve dilatation of the pupil is produced, the instillation of atropine produces a still further dilatation. In the lower cervical and upper dorsal region of the cord there is a so-called cilio-spinal centre, to which fibres pass from the anterior part of the floor of the aqueduct of Sylvius. The fibres leave with the first two dorsal nerves, pass in the rami communicantes to the cervical sympathetic, thence to the cavernous plexus, Gasserian ganglion, the ophthalmic division of the fifth nerve, and thence along the nasal branch to the long ciliary nerves to the eye. The pupil-dilating centre responds to every sensory stimulus, *e.g.* prick by a pin, pinching the skin, etc. Emotional states, *e.g.* fear, etc., also produce dilatation. This dilatation to stimuli takes place even if the cervical sympathetic be divided, and it is not improbable that there is a centre in the medulla oblongata. The condition known as *hippus* is one in which an oscillating condition of the pupil is present, contraction to a slight extent alternating with a similar slight dilatation.

Such are the reactions which occur in the healthy pupil. It has been already stated that there is no normal size for the pupil, for it varies in different individuals, depending probably in some degree on the difference in colour of the iris, and it also varies in the individual at different times in accordance with the degree of light to which it is exposed, the accommodative effect of the eye, and probably to some extent with the physical and mental states of the patient. But in spite of these so-to-speak normal, or non-morbid variations, unusual degrees of contraction—myosis—and of dilatation—mydriasis—and departures from the normal reactions to light and during accommodation, occur as the result of diseased conditions. From what has already been said, myosis, it will be understood, may occur from *irritation* of the centre for contraction of the pupil or of the afferent or efferent paths, or as a result of the paralysis of the mechanism on which dilatation depends. The former, *e.g.* occurs in irritative brain disease, in tobacco amblyopia, it is said, or as a result of habitual contraction in the course of daily work, as in watchmakers, etc. Such a pupil, although already contracted, will contract still further on exposure to bright light, and mydriatics will dilate it. Paralytic myosis is found most frequently as a result of disease in the cervical region, disease affecting the sympathetic path for dilatation. The contraction of the pupil so arising may be great, but the reaction of the pupil still remains. Mydriatics will only partly dilate it. It is only when higher structures are diseased

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that the pupil becomes quite inactive to light as well as contracted, but it may still retain its contractility with accommodation—a condition to be presently alluded to as the Argyll-Robertson pupil. Myosis may also result from excessive vascularity of the iris, as in iritis, or when a foreign body is present on the cornea. Mydriasis or dilatation of the pupil, the opposite condition to myosis, may also arise. That too may be irritative or paralytic—the former arises from irritation of the mechanism upon which dilatation depends, viz. the sympathetic chiefly, and it may be present as a result of irritative disease in the neck or in any part of the sympathetic path, in blood states which probably act as sympathetic irritants, *e.g.* anæmia and chlorosis, or it may arise by diminishing the vascularity of the iris, and it is also said, with doubtful truth, in conditions of increased intracranial pressure. Paralytic mydriasis, on the other hand, is the result of paralysis of the pupil-contracting centre and of interference with the path by which stimuli are transmitted to that centre. Thus disease of the third nerve nucleus or intraocular tumours may cause it. It has also been found in thrombosis of the cavernous sinus and in cases in which there has been pressure on the ciliary nerves. It also of course occurs in most cases of optic atrophy, and it occurs after excessive hæmorrhage.

One of the most important and significant modifications of the pupil occurring as a result of disease is that already alluded to as the Argyll-Robertson pupil, also known as reflex iridoplegia. In this condition there is often myosis, but this is not invariable. The essential of the condition is that the pupil while still reacting well during accommodation does not react at all—or so slightly as to have the same significance—to light. The pupils are frequently unequal, sometimes they are irregular in shape, *e.g.* oval instead of round. As a rule, when the Argyll-Robertson pupil is present it is so in both eyes. Occasionally, however, it is uniocular. It is a condition which usually occurs in association with tabes dorsalis; not infrequently it is also found in general paralysis, and exceptionally, in other cerebral and spinal diseases. It may be present without other obvious morbid conditions of the nervous system, but it is usually significant of at least a degenerative tendency in the nervous system. Its frequent association with diseased conditions of the nervous system, usually regarded as syphilitic or para-syphilitic, and its occurrence as an isolated phenomenon in patients with a syphilitic history, make it probable that this disease has a close causal connection with it.

Another morbid condition is that which occurs after diphtheria, and it is almost the converse of the Argyll-Robertson pupil, the condition, namely, in which there is paralysis of accommodation and consequent inability to read or see clearly at a near distance, while reaction to light is present. Rarely this is an isolated phenomenon; more commonly it is associated with other evidences of post-diphtheritic paralysis,

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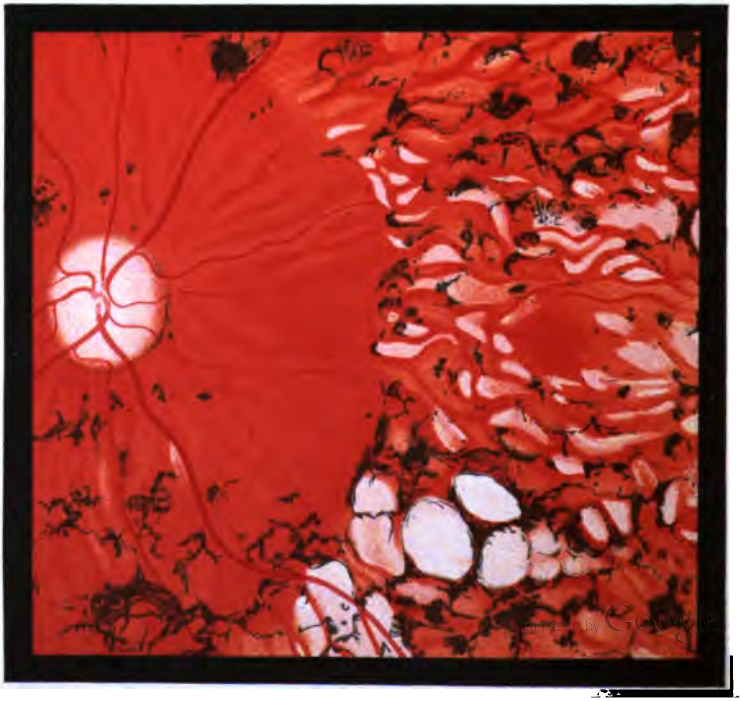
PLATE V.

FIG. 1.

FIG. 2.



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Illustrations of appearances of the Optic disc and Fundus oculi as seen with the ophthalmoscope. From *Ophthalmoscopic Atlas*, by Adams Frost, F.R.C.S.

PLATE V

FIG. 1.—Normal disc and fundus. The disc has a large physiological cup and a pale band at its circumference,—the connective tissue ring.

FIG. 2. — Disseminated choroidal atrophy from a case of congenital syphilis. Patches, white or pale in colour, are scattered over the fundus. The large ones are round or oval, the smaller ones irregular in outline. They have as a rule a ring of pigment round them, and the pigment tends to assume a reticular arrangement, with irregular meshes. In this condition the disc often assumes a papery whiteness, sometimes spoken of as waxy atrophy.

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PLATE VI

FIG. 3.—Papillitis (optic neuritis) with hæmorrhages on and near the disc. The veins are much engorged and the disc outline blurred, but the physiological cup is not filled in, as it so often is in such a condition. In this case the vision was not impaired and the visual fields were normal.

FIG. 4.—Simple gray atrophy of the disc (primary optic atrophy) from a case of tabes dorsalis. Such atrophy is sometimes spoken of as "tabetic," because the optic atrophy occurring in tabes dorsalis is nearly always of this variety. The disc outline is hard and clear, its surface a bluish pale gray, and the vessels are not, in this particular instance, reduced in size as they usually are. The absence of raggedness at the disc margin, and of white lines along the vessels, distinguish this condition from the atrophy following papillitis—the so-called post-neuritic or consecutive atrophy.

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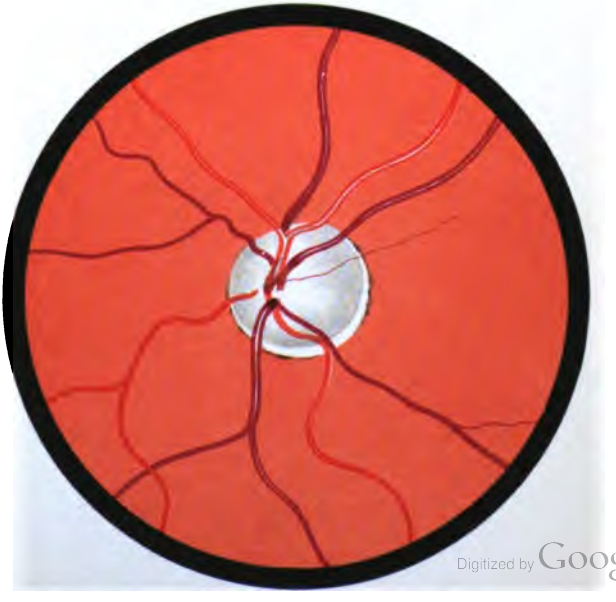
FIG. 3.

FIG. 4.



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such as affections of oculo-motor muscles, weakness of limbs, loss of the knee-jerk. The reaction to light is present unless the efferent limb of the light reflex is interfered with through the affection of the third nerve.

It has already been mentioned that inequality of pupils may be present with perfectly normal reaction, and probably in quite healthy persons. Inequality may also be present, as has been stated, with Argyll-Robertson pupil. Sometimes inequality of pupil depends upon interference with the sympathetic in the neck. Irritation on one side may cause a dilatation on that side, and this is usually associated with an increase in the size of the palpebral aperture. The pupil, however, on the affected side may be smaller, and a slight degree of ptosis may be present, and this would indicate sympathetic *paralysis* on this side.

Inequality of pupil and interference with ordinary reactions may also be the result of anterior or posterior synechiæ—evidences of old iritis.

It should also be remembered that operations on the iris, such as iridectomy, may give rise to irregularities in the pupil. The same also is true of a developmental defect, such as coloboma, in this structure.

Morbid Changes in the Fundus Oculi

Certain morbid affections of the ocular structures which show themselves in altered appearances visible in the fundus of the eye are of great importance in themselves and also as the indications of diseased affections elsewhere, usually of the nervous system, although other structures of the body may be those mainly concerned in the morbid process. The optic disc is probably the most significant part of the fundus, and changes indicative of disease may be visible in that structure while the rest of the fundus remains undisturbed. The changes which take place in the disc are—

(1) Those indicative of hyperæmia.

(2) Those indicative of inflammation—optic neuritis or papillitis.

(3) Those indicative of atrophy of the nerve—optic atrophy.

But other morbid conditions may be present, in which little or no change may appear in the disc, *e.g.* so-called retro-bulbar neuritis, in which the inflammation is behind the globe and only manifests itself later when the fibres involved have atrophied and so given rise to some change in the appearance of the disc. On the other hand, changes in the disc may succeed some morbid vascular process such as embolism, and may also be consecutive to some diseased condition of the retina and choroid. And there may be retinal changes such as hæmorrhages unassociated with visible morbid appearances in the optic nerve, and depending upon some blood state, such as anæmia, leucocythæmia, uræmia, etc.

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It will be well to consider first the changes which manifest themselves in an alteration in the appearance of the optic disc.

Hyperæmia of the disc.—There can be very little doubt that this is a term which has been used very loosely. It must be remembered that optic discs vary much in actual colour, and the appearance of the disc depends a good deal on the colour of the surrounding fundus. A red disc is not therefore necessarily either a hyperæmic one or a congested one. Of course if the observer has satisfied himself that the disc has become redder than it usually is, the change in colour is of the greatest significance, but much care must be exercised in describing any disc on the first examination as hyperæmic. On the other hand, such an appearance of hyperæmia in a disc formerly recognised as normal is of great importance as probably indicating the commencement of a true inflammation. In one kind of disc, viz. the disc of the hypermetropic eye, hyperæmia seems to be the rule. Occasionally the hyperæmia is so marked as to be indistinguishable, so far as ophthalmoscopic appearances go, from a slight degree of optic neuritis. The writer has known such cases in which even competent and careful observers have diagnosed optic neuritis. In one case, that of a boy under Mr. Marcus Gunn's care, such an error was not only excusable but almost inevitable. But Mr. Gunn had observed him continuously during several years without detecting any change in the appearance. As a rule, in these hypermetropic cases the physiological cup remains distinct.

Optic neuritis or papillitis (Pl. VI., Fig. 3). — Hyperæmia or congestion of the disc is also the initial condition in cases which are subsequently undoubted optic neuritis. Of course it is open to any one to say that such a condition is really optic neuritis, but a state of the disc can be detected in some of these cases in which, while it is evident that the disc is redder and the veins fuller than usual, it is still impossible to say that the condition is one of undoubted neuritis, and it is convenient to name such a condition congestion or hyperæmia. But in such cases, usually after the lapse at most of a few days, other signs are superadded. The surface of the disc becomes redder, its outline more indistinct—a fact which is more evident in the direct image than in the indirect, the veins become fuller, and there may be small hæmorrhages alongside them. Examination also shows that the surface of the disc is a little raised, and the vessels can be seen to dip over its edges. There are white lines along the vessels, and there may be a number of glistening white spots visible in the fundus, either arranged in a circular manner (asterisk) round the macula lutea, or arranged as a fan with its centre towards the disc. These in many cases are extremely like the similar appearance met with in albuminuric cases, and, according to Mr. Marcus Gunn, are the result of œdema and consequent wrinkling of the retina. The swelling of the

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disc may go on to an excessive degree until it may reach a height of as much as three millimetres; the hæmorrhages may become larger and more abundant, and besides the glistening white spots already alluded to there may be white woolly-looking patches, again resembling the condition found in albuminuric cases, probably the result of exudation. What is surprising is that in many of those cases in which the swelling may be considerable and hæmorrhages numerous, little or no interference with vision results. This was many years ago pointed out by Dr. Hughlings Jackson, and has been verified by numerous observers. In most cases in which the condition of neuritis persists, of course interference with the function of the nerve ultimately does supervene, but this often only after the inflammatory process has obviously passed into one of atrophy. All cases of optic neuritis do not go on to atrophy. In many the neuritis subsides, either as a result of medicinal treatment or of operative measures, sometimes it seems to do so spontaneously, and often when it has disappeared it is extremely difficult to say from the conditions left behind that there has been any inflammation, so complete is the subsidence of the morbid appearances. Occasionally some white lines along the vessels, or a somewhat ragged appearance of the disc outline, or slight obscuration or filling in of the physiological cup may be all that is left to mark the past existence of even a very acute optic neuritis. When, however, atrophy of the nerve results, this may be partial or complete, and there may be a corresponding impairment or a complete loss of vision. The disc gradually loses its redness and becomes white; the arteries are narrowed in their calibre, the veins long remaining engorged, and the outline of the disc by degrees becomes more defined, retaining for a considerable time, however, a certain woolliness in outline which is afterwards succeeded by the raggedness and irregularity of its margins already alluded to.

Such are the broad general features of optic neuritis as viewed through the ophthalmoscope. Several varieties might be described, the differences in their characteristics depending chiefly upon the acute or chronic character of the process, not necessarily the acute or chronic character of the morbid process underlying the neuritis. It is true that an acute process, *e.g.* a rapidly growing tumour, will naturally cause an acute neuritis; it does not, however, necessarily happen that a slowly growing or chronic tumour will cause a chronic neuritis. There is little doubt that a slowly growing tumour may exist for a long time without giving rise to neuritis, and when the neuritis does occur—if it is set up—it may be of an extremely acute or severe character. On the other hand, it may be slight in degree and exist for a long time as a slight chronic inflammation. When hæmorrhages form a prominent feature in the neuritis, when moreover these are large in size, more especially if they occur on the disc itself, where they are more uncommon than around its margin, the process is usually a very acute one and the

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swelling of the disc is great, and such a condition, as a rule, connotes an acute process underlying the neuritis. And large and numerous hæmorrhages are usually associated with much effusion and many glistening white spots, these being apparently the result of œdema of the retina.

It will now be convenient to consider *seriatim* the diseases in which optic neuritis is likely to occur, and the possible variations in the nature of the neuritis in so far as these may be significant of the main disease. It will be best to consider optic neuritis as it occurs :—

(1) In intracranial disease.

(2) In spinal disease.

(3) In morbid blood states.

In intracranial disease.—Meningitis is one of the diseases in which optic neuritis may occur. This may be traumatic, and of the neuritis which occurs in this form it may be said (and, indeed, this is true of the neuritis occurring in most forms of meningitis) that it is not often of a very intense character, but is apt to be comparatively slight so far as swelling and hæmorrhages and effusion are concerned. This is true even of the cases in which the meningitis becomes purulent; occasionally, however, a more intense inflammation may be present. The most common form of meningitis is tubercular meningitis, especially common, of course, in children. In most of these cases the neuritis is slight; indeed, it may not show itself by obvious changes in the fundus, and it is frequently a very late phenomenon. The occurrence of tubercles in the choroid must, however, be mentioned. These are not present in every case. Sometimes only one can be detected; occasionally larger numbers—as many as twelve it is said—may be present. They show themselves through the opthalmoscope as small yellowish-white patches, somewhat resembling patches of choroidal atrophy, but distinguished from those by the indefiniteness of their margin, by the absence of choroidal pigment round them, and by the concealment of the choroidal vessels. They vary in size from $\frac{1}{3}$ mm. to 2 or $2\frac{1}{2}$ mms., but occasionally several may coalesce to form a larger mass. Sometimes larger tubercles, the so-called "gray" tubercles, may be seen. These are usually visible in more chronic cases, and are sometimes discovered accidentally in the course of a routine examination. They have not the same serious significance which the others have, being more frequently the accompaniment of chronic tubercular processes, and they not infrequently disappear, leaving merely a scar to mark their former position.

The meningitis which results from ear disease is also sometimes the cause of neuritis. It must, however, be remembered that not infrequently this condition may be accompanied by the formation of cerebral or cerebellar abscess, and very intense optic neuritis occurring in the course of such a meningitis should suggest at least the possibility of the concurrent presence of abscess. If the optic neuritis is slight and not

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characterised by considerable swelling, the presence of abscess is not so probable.

Cerebrospinal meningitis may be marked by the presence of optic neuritis. It is not by any means invariably present either in the epidemic form or in the sporadic cases of uncertain etiology. It is occasionally present, however, and may lead to grave impairment of vision, which may persist after recovery in other respects has taken place.

In intracranial tumours the presence of optic neuritis is the rule. Not infrequently, however—and this is true especially of cases in which the tumour is a slowly growing one—a growth may be present for a long time without setting up neuritis. But even in those cases it not infrequently happens that when the symptoms become very severe, from the increased size of the tumour presumably, optic neuritis supervenes, and may in a very short time become intense. In cases in which the tumour is of very rapid growth neuritis usually occurs early in the history of the case. The presence or absence of optic neuritis and its degree of intensity do not furnish any really reliable information as to the nature or position of a growth. It is the writer's experience that most cases of cerebellar tumour give rise to very intense optic neuritis often with hæmorrhages and an abundance of white glistening patches, resembling those that occur so frequently in Bright's disease. It has been stated that tumours in the medulla oblongata do not as a rule give rise to optic neuritis. Of course tumours in this position cannot attain to very large dimensions without interfering with structures essential to life, and this may be the explanation in part at least of cases in which this absence of optic neuritis is true. The writer has seen two cases of tumour restricted to the medulla oblongata which were carefully and frequently observed up to the time of death, and in neither case was any optic neuritis ever present. In tumours of the pons, which are mostly of the infiltrating variety, giving rise to so-called hypertrophy of the pons, optic neuritis is usually present, but occasionally evidence of considerable interference with the function of pontine structures is present before optic neuritis occurs. It is not very common, however, for neuritis in such cases to lead to complete blindness. It is quite the opposite in cases of cerebellar tumour, in which sight is often completely lost. In the case of tumours of the hemispheres, optic neuritis usually occurs. It is probably not so frequent in cases in which the tumour grows from the dura mater, and is more likely to occur if a tumour is in the substance of the hemispheres than if it is growing from the surface. Large exostoses may occur growing from the inner table of the skull without setting up neuritis.

In a small number of cases there is a marked difference in the degree of the neuritis in the two eyes. In a much larger number there is a much slighter difference, although in the majority the neuritis is practically equal in the two eyes. Some observers have thought that in

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the cases in which there is a distinct difference the neuritis is more intense on the side on which the tumour is situated. Others have maintained quite a contrary view. In sixteen cases observed by the writer, in which the opthalmoscopic observations were made by Mr Marcus Gunn, and the swelling carefully and accurately measured, and in which the presence of tumour was confirmed by post-mortem examination, in eight cases the swelling was greater on the side of the lesion, in eight it was greater on the side opposite to the lesion. It is possible that the position of the tumour in reference to the optic nerves on the two sides may determine this difference. Thus a tumour in the frontal region on one side may cause greater pressure or a more intense neuritis on the same side, whereas a tumour in the occipital region may cause greater pressure or greater neuritis in the opposite optic nerve. This is merely a surmise, and there does not seem to be sufficient evidence at our disposal as yet to enable us to determine the cause of the greater intensity of the neuritis on one side. In a small number of cases of tumour the optic neuritis may be uniocular. In one case of this nature seen by the writer, the tumour was on the side in which the neuritis was present. But there is little doubt that in some cases of uniocular neuritis the neuritis is on the side opposite to the tumour, and we must regard these cases of one-sided optic neuritis as the extreme degree of a difference on the two sides, and as probably determined by the same causes which account for such a difference.

Abscess of the brain is a much more uncommon disease than tumour. As a rule optic neuritis is present. The most intense swelling the writer has seen, viz. 11 D, *i.e.* more than three millimetres, occurred in a case of cerebral abscess. On the other hand, some cases have been described in which no optic neuritis was present. Abscess is most common in the cerebellum and in the temporo-sphenoidal lobe, and in these positions is usually secondary to ear disease. But it may occur in any position. The presence or absence of neuritis does not seem to be of any significance in reference to the position of the abscess.

Optic neuritis sometimes occurs in the course of general paralysis of the insane. It is, however, by no means common. Some cases of widespread syphilitic change in the brain, closely resembling general paralysis in many points, but differing in being non-progressive and in yielding to anti-syphilitic treatment in some degree at least, have optic neuritis as one of their symptoms. This usually disappears under treatment. Occasionally in general paralysis the neuritis is succeeded by some degree of atrophy.

In spinal disease.—It may be said at once that in many of these cases the relation of the optic neuritis to the spinal disease is very obscure. In some cases of myelitis, whether the so-called idiopathic myelitis or that due to syphilis, optic neuritis has been found present, without any symptoms pointing to the existence of intracranial disease.

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It may possibly be the result in some cases of the toxic blood state which causes the myelitis. In some cases, especially in those associated with disease in the cervical region of the spine, it may be the result of an inflammatory affection spreading upwards or of some mechanical interference with the cerebrospinal fluid. It is occasionally present in cases of spinal caries. In one such case under the observation of the writer there were severe headaches, but without vomiting, and in one fundus there was an obsolete gray tubercle present. In another case, in which the disease was in the cervical spine, there was uniocular optic neuritis without any symptoms of intracranial disease. The spine was operated upon, the patient's paraplegia was cured, and the optic neuritis subsided. In another case with spinal symptoms—complete paralysis of all four limbs—there was double optic neuritis without any symptoms of intracranial disease. The patient subsequently died, and a small tumour was found high up in the cervical region of the cord, pressing upon it and accounting for the paralysis. The brain was most carefully examined and searched, but no intracranial disease was found. It is not improbable that in this case the neuritis was determined by some mechanical interference with the normal pressure of the cerebrospinal fluid.

In toxic blood states.—The next series of changes to be considered are those which occur in the optic nerve and fundus as a result of toxic blood states. Of these the most common and the most important are those which are associated with pathological states of the urine—albuminuria and glycosuria—or more correctly with the altered blood states giving rise to these.

The most common form of kidney disease in which neuro-retinitis occurs is the cirrhotic or granular contracted kidney. The percentage of cases in which ocular changes are found is variously stated by different observers. Probably 25 per cent is fairly near the mark. Similar changes, however, are also found in cases of the large pale kidney, and even in lardaceous disease.

The changes which occur in cirrhotic kidney disease affect several different structures. The nerve may be red, inflamed, and swollen—a condition really of optic neuritis. The vessels are characteristically altered. The arteries are often smaller than usual. Their calibre also varies at different points; the white glistening lines along them are distinct, and they themselves have an appearance of greater rigidity than usual, an appearance spoken of by Mr. Marcus Gunn as a "silver wire" appearance. They are also less transparent than usual, and at points at which they cross the veins the pressure is such that the veins are indented and the part of the vein distal to the artery is seen to be increased in calibre and engorged with blood, while the other part is narrowed and more empty-looking. Hæmorrhages may also be present—indeed in such a case they usually are. White, soft, woolly-

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looking patches also occur in various parts of the fundus, apparently patches of œdema, and there are frequently numbers of small white glistening spots in the fundus. These may be arranged around the macula in a concentric fashion, or may reach with a fan-like arrangement from near the outer side of the disc towards the macula. These spots are probably, according to the latest explanation, the result of a wrinkling of the retina. The same appearance is frequently seen in cases of intracranial tumour, especially if the neuritis is very intense, as it is apt to be in cerebellar cases, and many of these cases show an appearance practically indistinguishable from that seen in many cases of kidney disease. It is doubtful whether any of these appearances are due to degenerative changes, for in many cases in which they are present—especially in cases occurring during pregnancy, although not only in those—they completely disappear without leaving any definite traces behind them. Occasionally when a hæmorrhage has been present, a scar-like whitish patch may be left. Occasionally some pigment is present in such a case, but this is unusual—at least in any recognisable quantity.

In many of these cases the vision is definitely affected; in some the affection is slight or absent. Not uncommonly, however, in albuminuria, vision may be temporarily completely abolished—the result no doubt of uræmia affecting some of the nervous structures subserving vision. This may be the case when no changes in the fundus can be perceived with the ophthalmoscope, and is usually only temporary. In severe cases of albuminuric neuro-retinitis, however, the affection of the nerve may go on to complete atrophy.

Another morbid blood state in which pathological changes are visible with the ophthalmoscope is that of glycosuria. Changes are usually only found in cases in which the disease has been present for a long time, and usually only in patients past middle life. The proportion of cases of diabetes in which it occurs is thus, as we should expect, a very small one. Williamson in 100 cases found retinitis in 7, but in only 2 of those 7 was no albumen present in the urine, so that the albuminuria, it might be asserted, had to do with the retinitis. But although the changes in the retina in diabetics are very similar to those in albuminuria, there are certain broad distinctions which apply at least to characteristic cases. The white patches, as a rule, are larger in diabetes, and they are not arranged in the asterisk or fan-like manner so common, indeed almost invariable, in albuminuria. Hæmorrhages are numerous in diabetic cases, and unlike the flame-shaped hæmorrhages in albuminuria, they are usually rounded in shape. It is rare to find the optic papilla inflamed in diabetes—a condition very common in association with albuminuric retinitis.

Another condition found occasionally in diabetes is the so-called toxic amblyopia, in which there is a central scotoma for colour, exactly

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similar to what occurs from excessive tobacco-smoking. In many diabetics it is impossible to exclude the possibility of this as at least a factor in such cases, and it has been suggested that with a man whose resistance may be reduced by the diabetic blood state, a much smaller quantity of tobacco than usual may be sufficient to produce the symptoms. But in certain cases, especially in women with glycosuria in whom this toxic amblyopia has been present, it has been possible to exclude the influence of tobacco in evoking the symptoms. The toxic amblyopia, like that from tobacco, may be accompanied by pallor of the disc and even actual atrophy.

In certain cases of anæmia—not the pernicious variety, which will presently be referred to—optic neuritis has been found present. The significance of this is uncertain. It is possible that it is merely the toxic effect of a diseased blood condition on the optic nerve; it may be, as some have held, that it is the result of limited thrombosis of veins, similar to what we know occurs in the cavernous or other sinuses. It is important to know that it occurs, and that by its persistence it may gravely threaten vision. In many cases it completely disappears under the influence of treatment by iron and similar drugs; in a few it persists in spite of treatment, and in some it may, as has been said, result in complete loss of sight.

Pernicious anæmia is another blood state which frequently causes changes in the fundus. The most usual one is the occurrence of hæmorrhages with, or more usually without, changes in the optic nerve. These hæmorrhages are no doubt analogous to those which occur elsewhere in this disease, and probably result from the diseased condition of the vessels resulting from the impoverished or toxic state of the blood. A similar condition may result from leucocythæmia.

There is no doubt that occasionally optic neuritis is present in acute rheumatism. It is also said to occur in chorea and measles, and in these diseases it is probably the result of the blood state. It has also been found in scarlet fever, not always associated apparently in such cases with renal disease. In pyæmia also optic neuritis may be present, and it may be the result of the blood state alone, or of thrombosis in veins, or of local brain mischief resulting from the disease.

The condition known as retro-bulbar neuritis should be referred to, although, at its onset at least, no visible change is to be detected in the fundus. It is characterised by pain in the eye or eyes, and by the presence of a central scotoma for colours. After a time distinct pallor of the disc is usually to be detected, and this condition is due to interference with the function of the axial fibres of the nerve. It is likely that in some cases of disseminated sclerosis this is the process at work, but it occurs as an isolated phenomenon of obscure origin.

Atrophy of the optic nerve (Pl. VI., Fig. 4).—This may be either primary or consecutive to optic neuritis. The latter need scarcely be

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referred to so far as its etiology is concerned. It is characterised by a peculiar raggedness at the edge of the disc, by the obliteration or filling in of the physiological cup in the disc, and by the presence of white, glistening lines along the vessels. In the majority of cases these peculiarities are sufficiently distinct to enable the observer to conclude that a previous neuritis had existed. In some cases, however, it is exceedingly difficult to distinguish consecutive atrophy from that form which we now proceed to describe. Primary atrophy of the disc is characterised by the pallor of the disc, the diminished size of the vessels, and the fact that the physiological cup in the disc is not filled in, and the distinct openings in the lamina cribrosa may be quite visible at its base. It is usually spoken of as gray atrophy, or there is a bluish grayness about the disc, not the waxy whiteness which distinguishes certain cases of atrophy to be presently referred to. It occurs characteristically in certain cases of tabes dorsalis, but occasionally the atrophy in this disease may be consecutive to neuritis. It is also found in general paralysis of the insane, although in certain cases of this disease also the atrophy may not be primary, but may have been preceded by neuritis.

Injury to the nerve produced, *e.g.* by a blow or cut on the temple, may cause atrophy. Lesions at the optic chiasma, *e.g.* tumour of the pituitary body, do not as a rule produce neuritis. They cause atrophy of the optic nerve, and this lesion also gives rise to bi-temporal hemianopsia. Optic atrophy may also apparently result from loss of blood, *e.g.* from the stomach, bowels, uterus, etc. The exact reason for this is not easy to offer. The so-called waxy atrophy must also be mentioned. This is usually associated with choroidal changes—white patches surrounded by pigment—and is usually the result of late syphilis, congenital as a rule (Pl. V., Fig. 2). The optic atrophy which follows toxic amblyopia has been already alluded to. Optic atrophy is also present in a considerable proportion of cases of disseminated sclerosis. In this disease it is occasionally associated with a central scotoma for colours. The atrophic disc in this disease is as a rule of a papery whiteness, essentially different from the bluish-gray of tabetic atrophy. The vision may be very slightly impaired, even when the discs are very pale, and it may be stated as a general rule that whereas tabetic atrophy nearly always leads to complete blindness, the atrophy in disseminated sclerosis rarely if ever does. Occasionally, it is true, the sight of one eye may be completely lost, but it is very rare for both eyes to be so severely affected, although occasionally the impairment of vision may be very great. Sometimes in the atrophic disc of disseminated sclerosis there may be indications of a previous neuritis. This, however, is exceptional, and in most cases it may be said the pallor of the disc is no indication of the affection of vision. But even after all the forms of atrophy of more or less distinct etiology have been enumerated, a certain number remains of obscure and unknown origin, which we are forced to term

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idiopathic. Whether these are the first sign of some widespread disease of the nervous system, or whether they are the sole result of some obscure and unknown cause, is uncertain. In a few instances at least, in which the optic atrophy has remained for years an isolated phenomenon, symptoms of tabes or of general paralysis have subsequently appeared, but it is almost certain that this is not the case in all.

JAMES TAYLOR.

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THE MEDICAL APPLICATION OF ELECTRICITY

NATURE AND VARIETIES OF ELECTRICITY

The actual nature of electricity is not as yet definitely ascertained, although strong evidence has been adduced in support of the view that it is identical with the impalpable and all-pervading medium ("ether"), a particular form of wave-motion in which is sensibly obvious as light. Different conditions in this medium are loosely but conveniently known as forms or varieties of electricity. These are (1) electricity at rest, or **static electricity**; (2) **current or voltaic electricity**; (3) electricity (probably) in vortical motion or **magnetism**; and (4) **vibrant electricity.** Of this last several kinds are already known, and others no doubt await investigation. One, as we have already seen, manifests itself as light, another underlies those rays which were first systematically studied by Professor Röntgen.

1. **Static electricity.**—This is mainly, if not exclusively, evoked by physical processes, particularly that of friction, and can be demonstrated by the familiar experiment of rubbing a stick of sealing-wax upon the sleeve, or a glass rod upon a silk handkerchief. For medical purposes various kinds of electrical machines are employed, in which circular glass plates are caused to revolve in contact with brushes or rubbers; by an ingenious device a slight initial charge becomes enormously multiplied as the rotation continues. The charge is collected by means of fixed metal conductors furnished with teeth, and connected with the discharging knobs, either directly or through Leyden jars, which, acting as condensers, allow the production of a more powerful shock. The form of apparatus which is now in almost universal use is that devised by Mr. James Wimshurst.

2. **Current electricity.**—This is ordinarily the result of chemical changes, and requires the existence of a complete or *closed* circuit of conducting material. The essential element is known as a cell, wherein the current is generated by chemical action upon two dissimilar substances (usually but not invariably metals). These substances form the poles of the cell, which does not act unless they are connected externally as well. The energy of the apparatus is derived from the consumption or solution of one of the substances, which is conventionally termed the negative pole, the other, which remains intact, being the positive. The current passes in the cell from the negative to the positive pole, outside it from the positive to the negative, thus com-

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pleting the circuit. The term *anode* is applied to the spot at which the current enters anything, while the point at which it leaves is known as the *kathode*. Thus in a cell whose metals are copper and zinc the latter is the one consumed, and hence forms the negative pole; if a wire from the former be taken in the right, and from the latter in the left hand, the circuit will be completed through the body, the right hand forming the anode and the left the kathode.[1] Battery is the name given to a number of cells connected so as to multiply the effect. There are many kinds of cell, the advantages and disadvantages of which it is not proposed to discuss here. The batteries supplied by instrument-makers usually consist of cells containing acid, which can be renewed from time to time. For general medical use no form is more convenient or trustworthy than the Obach dry cell; as is the case with all sealed elements, this cannot be replenished when exhausted, but must be replaced. By means of suitable transformers, of which that devised by Woakes is perhaps the most popular, the electric lighting current can be adapted to many medical purposes.

Induced currents.—By interposing an induction coil in the circuit a battery may be made to give currents of great intensity but very brief duration. The apparatus consists of two coils of wire which have no connection with each other. The inner or primary is of somewhat coarse wire, and is wound round a core of soft iron (usually a bundle of wires); its two ends are joined to the two poles of the battery. The outer or secondary coil consists of an enormous length—often many miles—of extremely fine wire encircling a wooden bobbin, which can slide over the primary, the effect being greatest when it entirely encases the latter; its ends lead to two electrodes, by the application of which its therapeutic and other effects are produced. The passage of a current through the primary coil induces a powerful current in the secondary, but only at the instants when it is made and broken; by introducing a vibrating hammer, which rapidly makes and breaks the primary current, the secondary current, instead of being transient, is so frequently repeated as to give continuous physiological effects. The primary or ordinary cell current is usually spoken of as *galvanic*, the induced being called *faradic*. It must be remembered that the vibrating hammer is not an essential part of the faradic apparatus, which is, in some circumstances, of more value when used without it.

3. **Vortical electricity — magnetism.**—There is no satisfactory evidence of recognisable physiological effects of magnetism upon the animal body; therapeutically "animal magnetism" finds a place only in the armamentarium of quacks. The phenomena of magnetism only

[1] It should be noted, as regards the cell itself, that the *negative* pole is the one by which the current enters, and is hence the *anode*. Our ignorance as to the true nature of electrical processes is perforce clothed in a conventional nomenclature, which is sometimes confusing.

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interest the physician in so far as they are available in the mensuration of electric currents.

4. **Vibrant electricity.**—The form of this which is of medical value is that resulting from the repeated discharge of a powerful induced current through a vacuum (Crookes') tube or bulb. In this way rays arise whose power of penetration differs from that possessed by light rays, so that they can be used in the investigation of deep-lying structures and foreign bodies.

General Principles of Electro-Physiology

The physiological effects of the various "forms" of electricity are susceptible of such easy experimental investigation that their empirical use is quite unjustifiable; nevertheless the proper appreciation of the value of electrical methods in medicine has been greatly retarded by their unscrupulous abuse. It must, moreover, be remembered that the "moral" effect of treatment by electrical apparatus is very great, so that the utmost care must be exercised in recording successful cases. *Any* form of electrical treatment—which is in this respect but a variety of "suggestion"—may relieve "functional" symptoms, but organic disease must be dealt with along the lines established by physiology and pathology.

The influence of *static electrification* is probably felt by sensitive subjects during or before the occurrence of a thunderstorm; but it must be remarked that a similar feeling of depression, headachiness, etc., is often experienced when the barometer is low, whether there be "thunder in the air" or not. A statical or, indeed, any other kind of electrical discharge through the body will, of course, if sufficiently powerful, lead to instant death, a fact of which use has been made in connection with the infliction of the death penalty. The pathology of death from electricity was discussed in Vol. I. Exact and satisfactory experiments upon the effects of statical electrification have yet to be recorded.

The physiological consequences of *galvanic* currents are of two kinds, those occurring at the closure ("make") and opening ("break") of the circuit, and those observed during its continuance. The first partake of the nature of a "shock," and can be set aside by gradually raising the intensity from zero or diminishing it in the same manner. To the second, in the case of muscle or nerve, the name electrotonus has been applied; this effect is probably common to all forms of protoplasm, though we are deficient in the means of its demonstration. Certain it is that the passage of a weak current through living protoplasm (an amœba or a simple vegetable cell) increases the activity of pseudopodial or intracellular movement, while a more powerful one causes cessation of mobility and the retraction of the protoplasm into a

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ball; these effects are strictly comparable to those of increasing degrees of heat, and both physical agents will, if in excess, cause death. But the term electrotonus connotes more specialised consequences of the passage of a constant current through a muscle or nerve. These are of two kinds, physical (Dubois Reymond's) and physiological (Pflüger's). The former can be imitated by means of an appropriate model, composed of materials having different electrical "capacities," and is marked by the development of a current in the same direction as, and of equal duration with, that from the battery, and passing through the regions outside its poles as well as between them. Pflüger's electrotonus affects only living matter, and consists in alterations in its electrical excitability and conductivity. On making the current, and during its persistence, these, especially the former, are increased in the region of the kathode and diminished in that of the anode; on breaking it the effects are reversed. An interesting experiment of Biedermann's has demonstrated conclusively that on closing the circuit the excitation starts from the kathode; on opening it from the anode. These effects are complicated by the existence of "action" currents when the muscles contract ("currents of rest" are assumed to be non-existent in living uninjured tissues); in this case the muscle itself acts as a battery, the contracting portion being the negative pole and the current passing within the muscle from this to the relaxed region. This has also been described as the passage of a "wave of negativity" over the muscle contemporaneously with its contraction.

There is no doubt, moreover, that the passage through the tissues of constant currents insufficient to evoke physiological manifestations, such as contraction, can yet produce effects of the kind known as "anabolic," *i.e.* tending to build up the pabulum of tissue activity. This has been experimentally confirmed in human muscle, which has been shown to contract more vigorously, and to be less susceptible of fatigue after having been subjected to the action of a constant current insufficient in itself to cause contraction.

In addition to the physiological effects already mentioned, the chemical changes at the poles must also be noted. At the anode acid is produced, while at the kathode alkalisation occurs. As the blood and tissues are naturally somewhat alkaline, the anodic effect tends to be neutralised. But the kathodic is allowed full play; hence caustic and corrosive manifestations are exclusively or mainly confined to the latter pole. When these facts are combined with the changes in excitability it will be evident that with moderate currents a certain amount of irritation may be expected at the kathode, while the anode will exert a sedative influence.

The effects of the *faradic* or secondary current, particularly when the vibrating hammer is employed, are far more intense. The frequency with which the current is alternately opened and closed tends to abro-

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gate the difference between the poles, and, at the same time, leads to a summation of the stimulant effect. The resultant muscular contractions are hence tetanic in character instead of being simple twitches. The sensations produced are either sharp shocks or, with the interrupted current, a curious thrill, at first not unpleasant, but after a while attaining the quality of pain. In normal circumstances the application of electrodes over a muscle stimulates the intramuscular nerve terminals rather than the muscle fibres themselves; this is more evident in the case of the faradic current.

The physiological results of an electric current will obviously vary directly as its strength and the duration of its application; in addition to these factors two others must be reckoned with, the resistance of the body and the size of the electrodes. The resistance of the body is, as the name implies, the opposition which it offers to the passage of a current through it. The effect of such opposition is to transmute electrical force into other forms of energy. Thus the filament of an electrical lamp is the most resistant part of the circuit, and forms an obstacle to the current which results in a rise of temperature, so that it becomes first red and then white hot. It is hence plain that if strong or even moderate currents are to be medicinally employed without danger the resistance of the body must be very slight. It has been found that the resistance offered by internal structures is normally almost negligible, while that of the skin, particularly where this is thick, is considerable. Incautious procedures have hence led to painful and even serious cutaneous lesions, while in other cases the current has been allowed to waste its energy at the surface, and so to become useless for diagnostic or therapeutic purposes. By moistening the skin (best with warm salt water) its resistance may be sufficiently reduced to obviate these disadvantages. The influence of variations in the size of the electrodes may be illustrated by comparison with the flow of fluid through a tap. Given a certain pressure the force of this will be greater the smaller the jet, and similarly the intensity of electrical effects will vary inversely as the sectional area of the electrode through which the current is led in. For the production of shocks and intense local electrification it is hence advisable to use a small terminal, while milder and more continuous results follow the employment of a large electrode.

Finally, it must be noted that the electrolytic effects resulting from the contact of metal electrodes with the skin may be very painful; the terminals should therefore be wrapped in some moist material in which the chemical changes can be allowed to occur innocuously.

The progress of the electro-therapeutics has been attended by the creation of many new instruments, often of portentous size and complexity; it has not, as a rule, been deemed necessary to precede their application to morbid states in man by a scientific study of their

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physiological effects. Some are intended to combine different "forms" of electricity; all are highly efficacious when employed by their inventors. Reference will be made to the more generally accepted of them in the special sections which follow.

Electrical Mensuration

Clinically this is only of importance in connection with current electricity: statical electrification is merely measured by the length of spark which it gives in jumping through air to a conductor in connection with the earth; magnetic is of no therapeutic importance, and vibrant is at present without means of estimation.

The three important factors for our purpose are the *electromotive force*, the *resistance* opposed to it, and the *amount* of the current. The first of these is estimated in *volts*, the second in *ohms*, and the third in *ampères*. By Ohm's law the amount of current will be proportional to the electromotive force of the battery divided by the resistance which it encounters. It is measured by means of galvanometers, of which a horizontal pattern graduated in milliampères (thousandths of an ampère) is best for medical use, and regulated by introducing more or less resistance into the circuit. For the latter purpose a "resistance box" is used, in the most convenient form of which the current is made to pass through varying lengths of wire by merely turning a handle so as to bring a metal bar in contact with numbered studs; an exact knowledge of the amount of resistance can, if desired, be obtained by employing a voltmeter and calculating by Ohm's law from the readings given by this and the galvanometer. In the ordinary medical batteries the electromotive force is altered by bringing more or fewer cells into action, a process which is also effected by simply turning a handle; in using currents from the main the same object is attained by interposing various resistances, the electromotive force being of course constant. It may be here remarked that the current for electric lighting purposes is supplied by different companies in one of two forms, direct and alternating, and that while the former can be readily utilised for virtually any medical purpose, the latter is only conveniently applicable as a substitute for faradism.

The *dose* of current electricity varies of course according to the purpose for which it is required, but may be said ordinarily to lie between one and fifteen milliampères. From what we have already seen it is necessary with the larger doses to use larger electrodes in order to obviate unpleasant local effects. Where these effects are sought, as in electrolysis, a current of fifty milliampères, or even more, may be employed. A most important fact is that valuable therapeutic results can often be attained by means of currents of strength insufficient to cause muscular contraction or unpleasant sensations. Sir W. Gowers has

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rightly emphasised this point in connection with the treatment of infantile paralysis.

ELECTRO-DIAGNOSIS

At present only current and vibrant electricity are employed for diagnostic purposes. The physiological effects of the former have already been reviewed, and medically these are made much use of in muscle testing.

The reaction of degeneration.—Normally a muscle responds by contraction to a sufficient galvanic or faradic stimulus applied either directly or through the nerve. The nerve fibres are much more sensitive to faradism than the muscle fibres; there is indeed some evidence that the latter may be ordinarily stimulable only through the terminal nerve fibrils which they contain. When a nerve-muscle system (*i.e.* a neurone and its attached muscle fibres) is completely degenerate, or, in other words, dead, it can of course give no response to stimulation in any shape or form; in attaining this condition it may manifest either of two main sets of phenomena, dependent mainly upon variations in the rapidity of the lethal process. If the nutritional changes underlying this are slowly progressive, there is a steady and simultaneous fading away of both nervous and muscular irritability, and the response to stimulation dies out with equal gentleness in each case; the methods of electrical testing hence show only quantitative changes. But in more acute cases qualitative phenomena are superadded, giving a combination which is known as the reaction of degeneration (RD). In its most typical form this combination may be described under three heads. (i.) The nerve responds neither to faradism nor to galvanism. (ii.) The muscle responds to galvanism but not to faradism. This galvanic response is usually augmented in quantity, particularly in the early stages, and is curiously sluggish, tending to resemble the contraction of involuntary muscle. (iii.) There is an alteration in the relations of the galvanic response, as evidenced by muscular contraction, to different electrical conditions. Normally it is greatest when the kathode is applied to the muscle and the circuit closed (KCC). Next comes the anodal closing contraction (ACC), closely followed by the anodal opening contraction (AOC), the kathodal opening contraction requiring the greatest strength of current for its development. In a case of typical RD, ACC becomes as great as or even greater than KCC, a fact which can be readily demonstrated by using the smallest currents which will give a contraction, and putting a commutator or switch in the circuit so that the testing electrode can be made an anode or a kathode at will. The *modus operandi* is simple enough; one electrode is placed over the spine or the main nerve of the limb and the other applied to the muscles to be

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tested. Diagrams have been constructed—especially by Erb—of the points at which the maximum effects upon individual muscles can be evoked, the so-called "motor points." It is always of great importance to compare the corresponding muscles of the two sides, for which purpose the "indifferent" electrode, if placed over the spine, need not be moved. In testing the intrinsic muscles of the hands and feet it is best to place the two electrodes over the muscle, preferably on the extensor and flexor surfaces, so as to pass the current directly through it. The "active" electrode should be small—half an inch is a convenient diameter—and minimal currents should be employed. It is advisable also to have a galvanometer in the circuit to ensure that the current is not being wasted in overcoming the resistance of the skin; this can be conveniently lowered, as already stated, by moistening with salt solution.

In addition to the two chief types of deviation from normal electrical reactions, various intermediate forms are from time to time encountered. These are mainly conditioned by unequal affection of the individual fibres in a nerve-muscle system; the most common is one in which the nerve responds normally but the muscle reactions are of the degenerate type. Occasionally also in slight lesions the reaction stops short, so to speak, at a slight increase of irritability, often most marked to isolated faradic shocks. This condition may also sometimes be seen in chorea and paralysis agitans. The diagnostic importance of the reaction of degeneration is obvious. It can only indicate an affection, acute or subacute in nature, of the lower motor neurone. Very chronic lesions produce the gradual and contemporaneous failure of electrical response already mentioned. Disease of the upper neurone will evidently not give the reaction; perhaps if we could investigate the electrical conditions of the brain and cord we might find some evidence of such disease, but elementary physiological considerations forbid it from producing manifestations in accessible regions. Moreover, the reaction gives no information as to the pathological nature of the lesion; it does not tell us whether it starts from the nerve cell, as in infantile paralysis, or from the remote regions of low vitality, as in toxic neuritis. It should also be noted that the reaction is not at once fully established. Even in the acute degenerative process following section of a nerve the perfect type is not seen till about a week has elapsed, and with other causes its evolution is often slower. But its occurrence is indubitable evidence of a lesion of the lower motor neurone, which is at least of medium severity and of subacute rapidity of course.

The prognostic value of the reaction of degeneration is hardly less than the diagnostic. Other things—particularly the etiological factors—being equal, the prognosis of a case of paralysis from affection of the lower motor neurone is affected for the worse, as regards severity, duration, and prospect of ultimate recovery, by the presence of RD. Thus, in a case of acute anterior poliomyelitis, it is possible, at the end of a week

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or ten days, when both lower limbs are perhaps completely paralysed, to pick out by the reaction the muscle groups which are going to atrophy from those in which recovery is to be hoped for, or, in other words, to ascertain which neurones have had their nerve cells destroyed, and which only compressed, by the exudation. But in any case the prognosis is not so grave as with the slow failure of all response, or so hopeless as with its complete absence. For, in favourable circumstances and with appropriate treatment, as by the suture of a divided nerve, the degenerative reaction may pass away and the normal response return, voluntary power being usually re-established while electrical evidence of the lesion is still to be obtained.

Vibrant electricity in diagnosis.—The apparatus here employed consists of an induction coil giving a spark preferably of from 10 to 12 inches and excited either by a battery, or, less advantageously, by an alternating current from the main. Good results, especially in screen work, can also be obtained by the aid of a Wimshurst machine. The secondary current is passed through a Crookes' tube, best of the pattern devised by Mr. Jackson, the rays emitted by which possess the peculiar penetrative powers already referred to. The resulting appearances can either be recorded on a photographic plate or observed by means of a screen coated with barium platino-cyanide. The chief value of the method is surgical, particularly in the detection of foreign bodies and the determination of osseous and articular lesions, and in these respects much progress has been made since March 1896, when the writer published the first case of the use of these rays in England. Particular value attaches to the ingenious device by which Dr. Mackenzie Davidson, to whose zeal and skill the process owes so much, has rendered the exact site of a lesion or foreign body easily deducible from the photographs. The application of Röntgen's rays in medical diseases other than those affecting the joints has not as yet made much advance in England, chiefly owing to the unfortunate tendency on the part of some manipulators to see too much and to publish unverified diagnoses. Still Dr. Davidson has shown that in some instances renal calculi may be recognised, and it is possible, by the introduction of an œsophageal sound containing a leaden filament, to obtain valuable information as to the site of gastric and œsophageal strictures and neoplasms, as well as to diagnose and watch the effect of treatment upon dilatation of the stomach. And much is to be hoped in the diagnosis of thoracic and abdominal disease, particularly as regards the detection of deep-lying aneurysms and tumours.

ELECTRO-THERAPEUTICS

Great as is the value of electrical treatment, it is not, as some of its exponents claim, a panacea; it may in fact be said that there is no disease at present known for which it forms the

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sole therapeutic indication. It finds its legitimate and rational place in association with other lines of treatment—mechanical, as massage, medicinal, and dietetic. To effectively combine these requires both skill and experience, but the results will usually repay the care and patience demanded. The forms of electrification employed are mainly static and current; in a limited number of affections the vibrant variety also appears to be of some use. Attention must here again be drawn to the value of all kinds in the treatment of "functional disorders"; the main therapeutic factors seem to be the sight of the apparatus and the sensation of fairly sharp shocks. In connection with this it may be remarked that it is now common for lunatics, who would formerly have asserted themselves to be possessed by devils, to complain of being "electrified" by those whom their disease causes them to regard as enemies.

Static electricity.—This can be used either for general or local electrification. In the former case the patient is placed upon an insulated stool (one having glass legs varnished with shellac), and charged from one or other pole of the battery; the effects of this are described as "tonic" or "invigorating," and a not unpleasant temporary cutaneous glow is undoubtedly produced. Locally either the "electric breeze" or sparks may be used. The breeze is most conveniently applied by holding an electrode, consisting of a wire tassel connected to earth, at a distance of usually from a foot to a yard from a charged patient; a local sedative effect results. The consequences of these two methods closely resemble those following a cold bath and a local cold douche respectively. Sparks are best administered by passing a roller electrode over the particular region; it must be noted that the intensity of the effect is greater the thicker the layer of clothing through which the spark passes. Dr. Morton's method, in which the inner coats of two Leyden jars are attached to the prime conductors of the machine, and the electrodes led off from the outer coats (the so-called "static induction") is also of value. Both procedures produce more or less powerful cutaneous stimulation, which is of particular use in neuralgia and some "functional" disorders, but have no discernible advantage over the more convenient induction coil. Static machines require great care as to protection from damp and dust, and are perhaps of less utility in the climate of England than in other countries.

Current electricity.—This is also applicable to both general and local use. For the former the electric bath is most convenient; it is made of porcelain and has an electrode at either end, so that it can be charged by an induction coil, by the direct current from the mains, or best by the alternating or sinusoidal electric lighting current.[1] Great

[1] For more exact details the reader is referred to Dr. Hedley's work on *Hydro-Electric Methods in Medicine.*

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care must be taken to ascertain the strength of the current and the satisfactory working of the apparatus before immersing the patient. The effects are generally stimulant and comparable to those obtained by general statical electrification, or by the use of the currents of high potential. These latter are produced by charging (with a Wimshurst machine, a large induction coil, or an alternating main current) a couple of Leyden jars, whose outer coats are connected by a coil of wire. All these methods may be valuable adjuncts to, or even in some circumstances substitutes for, massage and other physical procedures. Local current electrification is used either for its stimulant or its destructive effects. For the latter fairly powerful faradic currents are employed, and the electrodes reduced to the smallest possible size (needles); by this means nævi can be cauterised and superfluous hairs removed; the therapeutic value of such applications to the urino-genital organs is now generally doubted.

The local employment of current electricity is of great and undoubted utility in certain diseases of the lower motor neurone, particularly acute anterior poliomyelitis and peripheral neuritis. Here there is no primary muscular lesion, but a defect in innervation, which is usually not wholly irreparable. The function of electricity is here to maintain muscular tone during the interval in which its normal nervous stimulus is wanting. If artificial assistance is not given, the restoration of functional activity to the neurone finds the muscle deficient in responsive power, and recovery is hence impeded and delayed. It is moreover known that each individual muscle is supplied by nerve cells situate in two or three successive segments of the cord; it is hence, perhaps, possible, when some of these are damaged, to heighten the susceptibility of the muscle to the lessened incitatory power, and so to minimise the defect. We have as yet no evidence of any favourable action of electricity upon the neurone itself; its value derives from its influence in the maintenance of muscular tone. The best results are obtained when the method recommended by Sir W. Gowers is adopted (see his *Diseases of the Nervous System*, vol. i., "Treatment of Acute Atrophic Paralysis"). The galvanic current is used of such strength as to cause muscular contraction without after-pain. The anode is placed at the upper end of the muscle, which is then gently stroked with the kathode. This process is systematically repeated in sittings of ten minutes or longer three times a week, or even more often, for usually six months or so; it is combined with massage and the other appropriate remedies, such as the administration of strychnine, and, in the case of toxic neuritis, the removal of the cause.

The sedative action of the galvanic anode can often be advantageously employed in the relief of pain, *e.g.* in neuralgia, and of excessive muscular irritability, as in some forms of incontinence of urine. Faradism, on the other hand, may be utilised by reason of its counter-

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irritant action; it is convenient in application, but its effects are unfortunately in general only temporary. In hysterical conditions, particularly where there is good reason to suspect malingering as a complication, the value of local faradisation is unquestionable. Hysterical aphonia yields immediately to the application of an interrupted current to the larynx, and the curative effects of the faradic wire brush in imitation fits are familiar to every one who has resided in a hospital.

It cannot be said that the therapeutic value of vibrant electricity is as yet definitely established. In the early days of its diagnostic employment, before the method was perfected, many cases of more or less severe dermatitis attested its powerful physiological action upon the skin. The Röntgen rays have accordingly been employed, with, it is stated, a considerable measure of success, in the removal of large tracts of superfluous hair. Attempts have also been made to ascertain whether internal diseases, such as phthisis, might not be ameliorated by their influence; it must, however, be admitted that the results hitherto obtained are not encouraging.

It is impossible here to go into more detail regarding the wide field of electro-therapeutics, nor is there space to describe the accessory medical uses of electricity, in connection with cauteries, cystoscopes, hot-air baths, etc., but, in conclusion, the following rules may be urged upon all those who wish to use electricity with benefit to their patients and themselves :—

(1) Test all the connections of your apparatus before use.

(2) Put everything away in place and clean after use.

(3) Whenever possible supervise each electrical application yourself.

(4) Try the effect of the current upon yourself before employing it upon your patient. If the latter is nervous, your own hand may often with advantage be used as an electrode.

(5) Cultivate "detachment" and a critical spirit in recording your results, and do not fail to take account of the other therapeutic agents which are concurrently employed.

BERTRAM ABRAHAMS.

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